Dissertation On

"A CLINICAL STUDY OF CEREBRAL VENOUS THROMBOSIS"

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CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICAL STUDY OF CEREBRAL VENOUS THROMBOSIS"** submitted by **DR.B. PRIYADARSINI,** appearing for Part II M.D., branch I General Medicine Degree examination in March 2011 is a bonafide record of work done by her under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, TamilNadu, India.

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DECLARATION

I solemnly declare that the dissertation titled **"A CLINICAL STUDY OF CEREBRAL VENOUS THROMBOSIS"** is done by me at Madras Medical College & Govt. General Hospital, Chennai during 2008-2011 under the guidance and supervision of **Prof. E.DHANDAPANI, M.D.,**

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CONTENTS

LIST OF FIGURES

LIST OF ABBREVIATIONS

ADC	→	APPARENT DIFFUSION COEFFICIENT
ANA	→	ANTENUCLEAR ANTIBODIES
APLA	→	ANTIPHOSPHOLIPID ANTIBODIES
CSF	→	CEREBROSPINAL FLUID
CT	→	COMPUTED TOMOGRAPHY
CVT	→	CEREBRAL VENOUS SINUS THROMBOSIS
DWI	→	DIFFUSION WEIGHTED IMAGING
EGG	→	ELECTROENCEPHALOGRAM
ESR	→	ERYTHROCYTE SEDIMENTATION RATE
GTCS	→	GENERALIZED TONIC CLONIC SEIZURE
HB	→	HEMOGLOBIN
ICP	→	INTRACRANIAL PRESSURE
INR	→	INTERNATIONAL NORMALIZED RATIO
LP	→	LUMBAR PUNCTURE
LS	→	LATERAL SINUS
MRV	→	MAGNETIC RESONANCE VENOGRAPHY
PT	→	PROTHROMBIN TIME
SS	→	SIGMOID SINUS
SSS	→	SUPERIOR SAGITTAL SINUS

INTRODUCTION

INTRODUCTION

Cerebral venous sinus thrombosis (CVT) has been recognized since the early part of the nineteenth century but still remains a diagnostic and therapeutic challenge for the clinician given the varying and often misleading clinical presentation of this condition. It forms a distinct subgroup of cerebrovascular disease and is one of the commonest causes of stroke in India.

Though earlier studies have reported higher mortality, recent studies have reported lesser mortality due to earlier diagnosis, increased awareness and management. Cross et al.[1] noted "usually recovery is rapid and complete, if the patient survives acute episode". Three fourth of cases of cerebral thrombosis in pregnancy and puerperium reported by him, survived with good recovery. Although it may present with a variety of signs and symptoms, headache is the most frequent and often the earliest manifestation[2].

The diagnosis of cerebral venous sinus thrombosis requires high index of suspicion. CT brain may show direct or indirect signs of cerebral venous thrombosis. It may be normal in 10% of patients[3]. In such cases advanced neurological diagnostic like magnetic resonance imaging with

venography is necessary to confirm cerebral venous thrombosis, but it is not always readily available in many hospitals. It has been found that early diagnosis of cerebral venous thrombosis is essential because early treatment may prevent morbidity and may even be life saving. Cerebral sinus venous thrombosis is considered to be a medical emergency, [4] mode of onset highly variable, and spectrum of its clinical manifestations is extremely wide.

Management of CVT with anticoagulants especially heparin was a controversy in earlier studies, but recent studies have proved heparin to be both effective and safe even with hemorrhagic infarcts. Heparin administration is the standard treatment for CVT[5]. Although lysis of the sinus thrombus does not occur with systemic anticoagulation, prevention of new thrombus formation is essential.

The goal of heparin therapy is to halt clot proliferation and prevent worsening of clinical symptoms[6] (Bousser & Russell, 1997; chow et al., 2000). Heparin is generally continued until the patient stabilizes (Bousser & Russell). However, intracranial hemorrhage is a significant potential consequence of this therapy[7], and the benefits of anticoagulation must be carefully weighed against the risks.

Long-term medical therapy for CVT patients includes systemic anticoagulation with warfarin for an average of 6 months (cakmak et al., 2003). Fortunately, the risk of CVT recurrence during subsequent pregnancies is low (mehraein et al., 2003), and patients are not routinely counseled against future pregnancies.

AIM AND OBJECTIVE

AIM AND OBJECTIVE

I. To analyse the commonest clinical modes of presentation

II. To find out the possible etiology

III. To analyse the topography of involved venous sinuses in Magnetic resonance venogram.

IV. To evaluate clinical outcome.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Strokes resulting from cerebral venous thrombosis are common and have a distinct clinical profile, usually affect young persons, predominantly women and carry a significant mortality if not adequately treated. The term "primary" or "idiopathic" cerebral venous thrombosis is used when no specific etiological factor is evident. "Secondary" sinovenous thrombosis results from a variety of causes that include injury, infection, hematological disturbances, dehydration, etc. In India CVT in the early puerperal period is very common, 10-12 times higher than in the west.[8,9,10]

History

Ribes[11] first reported primary venous sinus thrombosis in 1825. He described in a 45 year old male, the clinical and autopsy spectrum of superior sagital thrombosis. In 1828, John Aberocombei[12] of Scotland described this entity in a 24-year old lady who died from this disease. Autopsy showed ischemic and hemorrhagic infarcts with thrombosed and sclerosed cortical veins. Kalbag and Woolf[13], Purdon martin, Sir Charles symmonds and others gave a precise clinical description of CVT after 1940.Several large series with confirmation of diagnosis by angiograms,

surgical exploration, autopsy and recently with CT and MRI studies have been reported from the Indian subcontinent.[8,9,13]

EPIDEMIOLOGY

The true incidence of CVT is unknown. Ehlers and Courville[14] found only superior sagittal sinus thrombosis in a series of 12,500 autopsies. Towbin found CVT in 9% of 182 consecutive autopsies[15]. However, with more recent studies of large clinical series, the true incidence of CVT is probably considerably higher than that thought from autopsy series. Exact figures however remain elusive. CVT constitutes 10 -15 % of stroke according to Indian studies.[8, 9, 13] Especially in the young and in pre-menopausal women. People of all age groups may be affected but there is slight preponderance in young women because of specific causes like oral contraceptives, pregnancy and puerperium.[16]

Puerperal CVT has been reported to account for up to 15-20% of young stroke.[12] It is the commonest cause of stroke in young women in India. Fifty percent of strokes in Indian women are related to pregnancy and puerperium and 95.5% of these are due to CVT[8.] Prevalence of postpartum CVT in India is 4.5/1000/year [8,9] In Indian population, multiparas are affected more than primipara in proportion of 4:112 in a study sample of 230 cases of CVT seen over a period of 8 years at

National Institute of Mental health and neuro sciences (Nimhans), bangalore, 200 cases were in the puerperal period. It has been estimated that prevalence rates of CVT in developing countries are approximately 10 times more than in developed countries. Currently aseptic CVT has replaced septic CVT as the commonest cause.

PROGNOSIS

CVT associated with pregnancy is reported to have a better prognosis than CVT arising from other causes, with a mortality rate of approximately 10% (Van der stege et. Al., 1997).[17] Researchers have estimated all-cause CVT mortality as 10%-30% (baker et. Al., 2001; chow et al., 2000). In a retrospective review of 170 peripartum patients with CVT, Lanska and Kryscio (2000) reported that more than 93% of patients were discharged home, and there were no deaths in their series. Although many CVT patients experience some persistent neurologic deficits, most will return to their previous level of function.

Relevant venous anatomy

The cerebral venous system consists of

(1) dural venous sinuses

(2) cerebral veins

The cerebral veins that empty into dural sinuses, which then drain into the two internal jugular veins.

The dural venous sinuses

The dural venous sinuses are valve less, transloculated venous blood channels and may be divided into a superior group related to the vault and a basal group found at the skull base. The sagittal, transverse and straight sinuses are the main components of the superior group. The basal group comprises of cavernous, petrosal and sphenoparietal sinuses. The dural sinuses most commonly thrombosed are the superior sagittal sinus (SSS), lateral sinus and cavernous sinus.

Superficial cortical veins drain into the superior sagittal sinus against the blood flow in the sinus, thus causing turbulence in the blood stream that is further aggravated by the presence of fibrous septa in the inferior angle of sinus. This explains the greater prevalence of superior sagittal sinus thrombosis Intercommunication between the various venous sinuses provide alternative routes by which blood from dural sinuses may flow if normal drainage is blocked.

Superior sagittal sinus (SSS)

SSS lies in the anterior border of falx cerebri and runs from the foramen caecum to the occipital protuberance, where it joins with straight sinus lateral and torcular herophili. The SSS is triangular in cross section, increases in size from before backwards. In the majority of individuals, most of its flow is directed to the right transverse sinus with the straight sinus draining to the left transverse sinus.Cortical veins enter perpendicular to the SSS anteriorly but the angle becomes shallower more posteriorly as the veins entering against the direction of flow. SSS and other sinuses play a major role in CSF circulation because they contain most of the arachnoid villi and granulations (pacchionian bodies) in which much of the CSF absorption takes place.

2. Inferior sagittal sinus

Situated in the posterior 2/3 of falx cerebri. It joins the great cerebral vein to form the straight sinus that eventually ends in the left transverse sinus.

3. Lateral sinuses (LS)

The lateral sinuses extend from the torcular herophili to the jugular bulbs and consist of the transverse and sigmoid portions. They

drain blood from the cerebellum, brain stem and posterior portion of cerebral hemispheres.

Transverse sinus(TS): Begins at the confluence of sinus (torcula heterophili) to pass laterally and forward in the margin of tentorium cerebelli to the postero inferior angle of the parietal bone and passes down as a sigmoid sinus. It receives blood from the superior petrosal sinus, inferior cerebral veins, and inferior cerebellar veins.The right is usually 'dominant' and larger than on the left, receiving almost the entire output of the superior sagittal sinus. The sinus on one side can be poorly developed or even absent.

Sigmoid sinus(SS): It is the direct continuation of the transverse sinus and is 's' shaped. It extends from the postero inferior angle of the parietal bone to the posterior part of the jugular foramen where it continues as the superior bulb of internal jugular vein. Its tributaries are the mastoid veins, cerebellar veins, internal auditory vein.

Straight sinus(STS): The straight sinus lies at the junction of the falx and the tentorium and the torcular herophilius where the straight, transverse and SSS meet. Numerous LS anatomic variations may be misinterpreted in sinus occlusions on angiography.

In hacker's study, transverse portions were not visualised on ipsilateral carotid angiograms in 14% of cases on left side and 33% on right side, whereas sigmoid portions, which may be directly injected via cerebral veins, failed to fill in 4% of cases on left side and were always demonstrated on right.

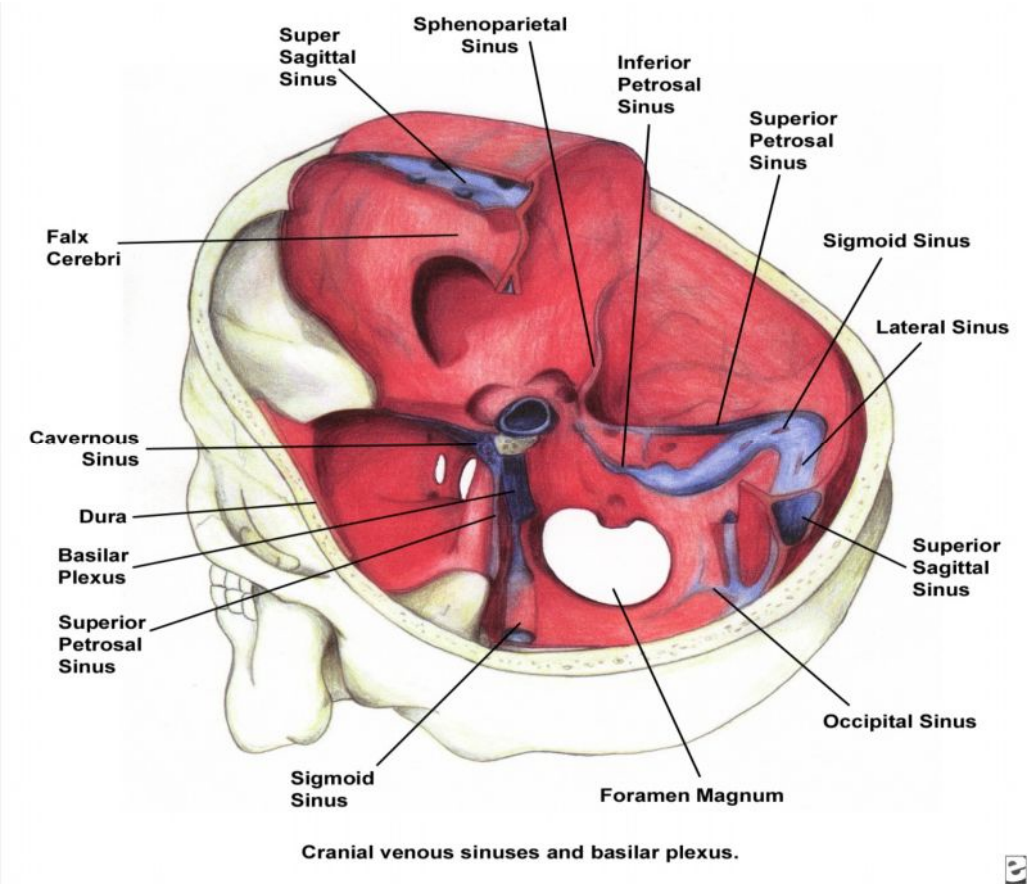
4. Cavernous sinus

This sinus drains venous blood from the orbits through the ophthalmic veins and from anterior part of the base of the brain via the sphenoparietal sinus and middle cerebral veins. They empty into both superior and inferior petrosal sinuses and ultimately into internal jugular vein. Because of their situation, cavernous sinuses are often thrombosed in relation to infections of face or sphenoid sinusitis. In contrast to other sites, infection is the leading cause of cavernous sinus thrombosis[18].

5. Petrosal and sphenoparietal sinuses

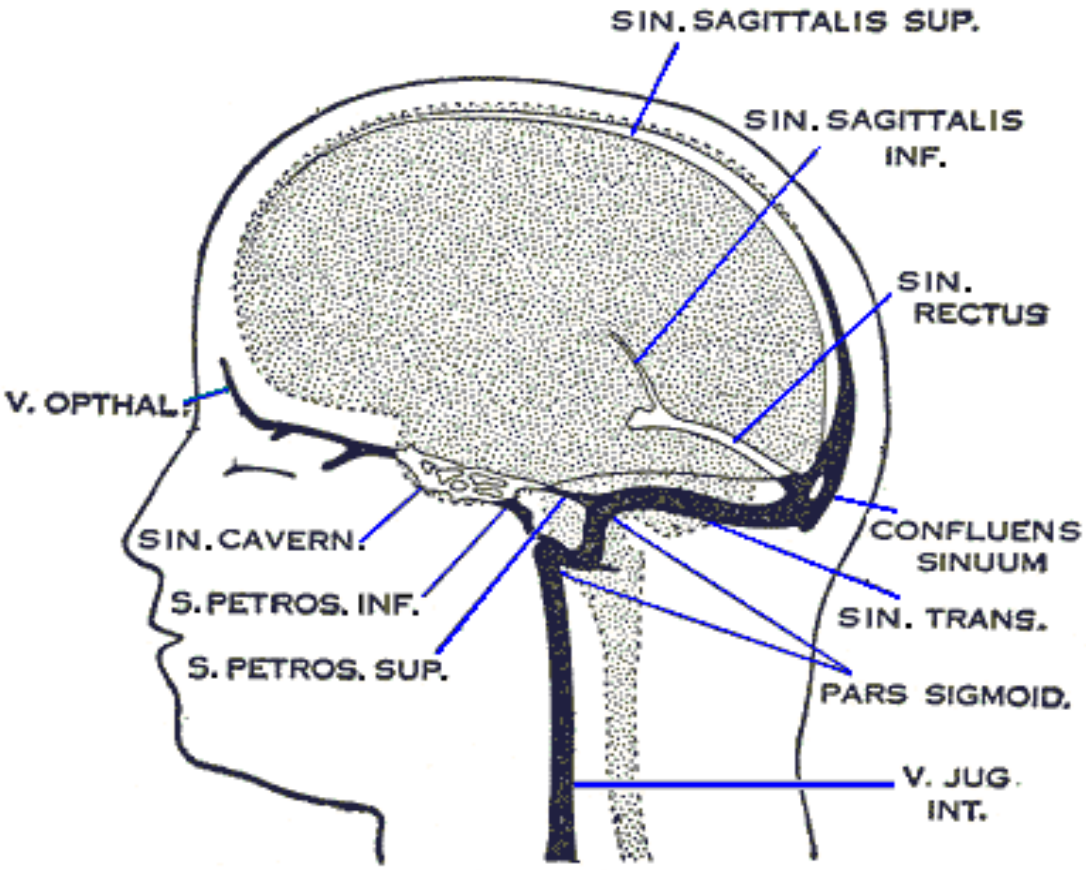
The superior petrosal sinuses are situated at the junction of the tentorium and the petrous bone. They drain to transverse sinuses. The inferior petrosal sinuses lie between the clivus and petrous apex and run medial to the superior sinus joining the jugular bulb. The sphenoparietal sinus is a medial extension of the sylvian vein and courses around the greater sphenoid wing.

FIG -1 SINUSES AT THE BASE OF THE SKULL



Cranial venous sinuses and basilar plexus.

FIG-2 SAGITTAL SECTION OF THE SKULL, SHOWING THE SINUSES OF THE DURA



The supra tentorial venous system:

Cerebral veins:

Three groups of veins that draw blood supply from the brain are:

1 Superficial cerebral, 2. Deep cerebral and 3. Veins of post fossae.

1. Superficial cerebral veins

Some of the cortical veins the frontal, parietal and occipital superior cerebral veins drain the cortex, which ascends to the SSS whereas others, mainly the middle cerebral veins drain into the cavernous sinuses. Troland's greater anastomotic vein connects the SSS to middle cerebral veins, which are further connected to LS by the vein of labbe.

The cortical veins present some peculiarities that are important to know to understand some of the clinical features of CVT –They have thin walls, no muscle fibres and no valves. These features allow for dilatation and reversal of the direction of blood flow when sinuses into which they drain are occluded. They are linked by numerous anastomoses, allowing development of collateral circulation (angiographically visible as corkscrew vessels). This probably explains the good prognosis of the venous thrombosis.

2. Deep cerebral veins

The internal cerebral and basal veins both join to form the great vein of galen, which continues as straight sinus which drains blood from the deep white matter of the Cerebral hemispheres and from basal ganglia. In contrast to the superficial system, the deep system anatomy is constant and is always visualized on angiography, so that thrombosis is easily recognized.[19] They are choroidal veins, terminal vein, and basal vein of Rosenthal.

3. Veins of posterior fossa

There are three groups:

(i) Superior veins draining into the galenic system.

(ii) Anterior veins draining into petrosal sinuses and

(iii) Posterior veins draining into the torcular or neighbouring SS and LS.

They are variable in course and angiographic diagnosis of their occlusion is extremely difficult.

Causes of CVT

In the pre-antibiotic era, infection was commonly associated with CVT. But now it has become less common, various causative factors implicated are infectious, inflammatory diseases, neoplasm,. coagulation abnormalities, pregnancy and endocrine conditions.[3, 6, 20.] Puerperium is associated with a six fold increase in risk of venous thromboembolism. Even in developed countries, despite extensive investigations, in 25% cases, no cause is found.

Table 1: CAUSES OF CEREBRAL VENOUS THROMBOSIS

A. Septic dural sinus thrombosis

- Local septic trauma
- Intracranial infections: abscess, empyema and meningitis
- Others -Otitis ,Sinusitis,Tonsillitis ,Stomatitis
- Systemic bacterial (typhoid, TB, septicemia, endocarditis)
- Viral (measles virus, hepatitis viruses, herpes simplex virus, HIV, cytomegalovirus)
- Parasitic (malaria, trichinosis)
- Fungal (aspergillosis)

B. Non-septic dural sinus thrombosis

- Hemodynamic states
  - Dehydration
  - Fever
  - Cardiac failure

- Hematological disorders -polycythemia vera
  - Secondary polycythaemia
  - Disseminated intravascular coagulation
  - Sickle cell anemia and trait
  - Cryoglobulinemia
  - Paroxysmal and nocturnal hemoglobinuria
  - Thrombocytosis
  - Severe anemia
  - Antithrombin-III deficiency
  - Protein C & S deficiency

- Hormonal dysfunction
  - Oral contraceptive use
  - Pregnancy and puerperium
  - Androgens

- Trauma
  - Penetrating and non-penetrating head injuries
- Surgery
  - Cardiac pacemakers
  - Jugular venous catheters
- Metabolic disorders
  - Homocystinuria
  - Osteopetrosis
  - Diabetes mellitus
- Neoplasia
  - Meningioma
  - Metastasis (usually hematogenous)
- Inflammatory disorders
  - Anti phospholipid antibody syndrome
  - Behcet's disease
  - Sarcoidosis
  - SLE
  - Wegener's granulomatosis
  - Polyarteritis nodosa
  - Inflammatory bowel disease

- Ulcerative colitis
- Chron's disease
- Cogan syndrome

❖ Vascular disorders

- Arterio-venous malformation
- Arterial occlusions
- Sturge weber syndrome

Pathogenesis of cerebral venous thrombosis

Various theories have been advocated for the pathogenesis of the cerebral venous thrombosis. The main factors incriminated are:

1. Infective theory
2. Embolism
3. Local endothelial damage
4. Hypercoagulability

1. Infective theory

The septic thrombosis is one where a purulent infection occurs with attendant Thrombophlebitis. The classic examples of such a condition are cavernous sinus thrombosis following facial infection and lateral sinus thrombosis following otitis media. In Daif a et al.[21] studied 40 cases of

CVT, infection was the cause of CVT in 7% of cases, whereas it was the cause in 16% and 17% of cases reported by Bousser et al[6] ,Shell and Rathe, [22] respectively.

2. Martin-Batson theory of embolic thrombosis

In understanding the pathogenesis of puerperal venous thrombosis, the studies of Batson (1940) and extension of the results of that study by Martin (1941) are milestones. Batson (1940) in experimental work on monkey and human cadavers showed that pelvic veins anastomose with cerebral plexus of veins, positive proof of functional conduit in live patients has not been shown. Based on these data martin (1941) argued that under circumstances of increased intra-abdominal pressure, the thrombi from parturient mother could pass into intracranial sinuses via the vertebral plexus. Once the thrombus reaches the sagittal sinus, where blood flow is slow, it acts as a nidus for further thrombosis[19].This theory fails to explain

(i) The basic mechanism of puerperal thrombosis.

(ii) The fact that SSS is most frequently involved, although vertebral plexus of veins communicates with the occipital and petrosal sinuses and not SSS.

(iii) Delayed onset of symptoms.

3. Kendall's theory of local damage

Patho physiologically, there are important differences between arterial and venous thrombosis. CVT has been described as a continuing process in which the balance of prothrombotic and thrombolytic processes is disturbed, leading to progression of the venous thrombus in time[24].This slow growth of thrombus and the good collateralization of the venous vessels probably explain the usually gradual onset of symptoms, frequently over weeks and months. Sudden onset however has been described.

Kendall[23] was among the first to suggest that etiology of puerperal CVT. It might be found by considering those aspects of the disorder that produce **Virchow's classic triad:** 1) stasis of blood flow; 2) vascular endothelial damage; and 3) a hypercoagulable state. If a careful search is made, each of these conditions for the generation of thrombosis may exist in the pregnant patient, in particular one who presents with toxemia or dehydration.

4. Stasis

Bailey (1971) noted that in many pathologically proven SSS thrombosis, the thrombus was oldest in the middle fifth of the sinus and

in some cases only the central portion was involved suggesting involvement of certain local factors. In this region the superior cerebral veins enter the sinus in a direction opposite to the direction of blood flow in the sinus. Also there is sudden widening of the sinus at this point and may contribute to the localisation.

5. Theory of hypercoagulability

The hemostatic balance changes in the direction of hypercoagulability, thus decreasing bleeding complications in connection with delivery. The most important initial factor for acute hemostasis at delivery is, however, uterine muscle contractions, which interrupt blood flow.

Increased endogenous thrombin generation, acquired protein C resistance, slightly decreased activated partial thromboplastin time (aPTT) and increased prothrombin complex level (PT) measured as INR of less than 0.9, activation of platelets, release of beta thromboglobulin and platelet factor 4 and increased thrombomodulin levels have been reported in normal pregnancy. Most blood coagulation factors and fibrinogen increase during pregnancy. Factor XI is the only blood coagulation factor that decreases during Pregnancy.

Fibrinolytic capacity is diminished during pregnancy, mainly because of markedly increased levels of plasminogen activator inhibitor 1 (PAI-1) from Endothelial cells and plasminogen activator inhibitor-2 (PAI-2) from the placenta. The total hemostatic balance has been studied by analysis of prothrombin fragment 1 and 2, thrombin-antithrombin complex, fibrinopeptide a, soluble fibrin, D-dimer and plasmin-antiplasmin complex. There is activation of blood coagulation and a simultaneous increase in fibrinolysis without signs of organ dysfunction during normal pregnancy. These changes increase as pregnancy progresses.

Fibrinolysis improves and increases fast following childbirth and expulsion of the placenta, resulting in increased D-dimer levels. These changes are self limiting at normal delivery. The hemostatic changes normalize after delivery within 4 to 6 weeks. Platelet count and free protein [8], however can be abnormal for a longer Period. [27-32]

Pathology

Hemorrhagic infarction occurs in approximately 10-50% of cases, principally affecting the cortex and adjacent white matter.[24, 33] This is

thought primarily due to elevated venous and capillary pressure caused by the persistence of thrombus.

Clinical features of cerebral venous thrombosis

Clinical features of CVT are very variable and depend on the site, extent and rate of thrombosis. If the symptoms are restricted to the dural sinuses, it may present with symptoms of raised intracranial tension. If the cortical veins are involved, focal deficits and seizures may be the presenting features.[34]

SYMPTOMS AND SIGNS

Headache

This is the most prominent and frequently presenting symptom in 70-80% of the patients.[35] There are no specific features. It may be acute, like a thunderclap headache, [36] or sub acute or chronic. In most of the cases, it is associated with nausea, vomiting and other neurological symptoms. It is probably caused by leakage of blood to the surface stimulating pain-sensitive fibers in the Dura or due to rise of intracranial pressure[37].

Isolated headache can be the only manifestation of CVT[35]. It is often unilateral, ipsilateral to lateral sinus thrombosis. Whether it represent a benign form of CVT that would have spontaneously recovered or the onset of a thrombosis stopped by heparin remains unknown but this uncertainty justifies early diagnosis and urgent heparin treatment.

Fever

This is often present with septic CVT or those secondary to systemic infections. Although not mentioned as an important feature in two large international Studies[38], it is frequently seen in Indian patients[41, 42]. Pyrexia is noted in 15-60% of such cases, does not necessarily imply infection and is probably caused by an aseptic thrombotic process.[43, 44] Superior sagittal sinus (SSS) thrombosis in infants and children may present only with fever and convulsions which may be confused for febrile seizures.

Seizures

These occur more frequently in CVT than in arterial stroke. They are very common and often begins early, may be focal or generalized and present in 50% of cases. Their early appearance is a sign of bad prognosis[45]. The seizures may be recurrent and may be followed by

aphasia and hemiparesis. Seizures may be focal, modal or generalized, and are commonly due to 'irritation' of the cortex when there is a hemorrhagic venous infarct. Status epilepticus may follow in some cases and at times, it is the presenting feature. It's incidence varies from as low as 3.5% to as high as 42% in two Indian series.[47,48]

Focal deficits and impaired sensorium

Focal signs vary with extent and site of thrombosis. They may be motor or sensory, unilateral or bilateral, and fluctuating. They are seen in approximately 60% of Cases.[49] Altered sensorium is usually due to raised intracranial pressure (ICP) which is often preceded by headache and severe focal deficits. Although moderate drowsiness is usually reversible with therapy, progressive worsening of sensorium is associated with a poor prognosis. Aphasia, mutism and cerebellar infarcts are also described.

Cranial nerve palsies are uncommon; but a total ophthalmoplegia is seen with cavernous sinus thrombosis and ninth and tenth nerve palsies with thrombosis of the internal jugular vein.[49] Hemiparesis usually with facial sparing, as facial area is drained by slyvian vein. The lower limb is affected more often than upper limbs. In some cases of superior saggital

sinus the contralateral cortical veins may be involved giving rise to contralateral paraparesis or paraplegia.

Pappiledema

Thrombosis of the dural venous sinuses leads to an increase in venous pressure and alters the pressure gradient leading to poor CSF absorption through the pacchionian granulations. Hence there is rise of ICP and pappiledema. The incidence of pappiledema is variable and depends on the aetiology, rate and site of venous thrombosis. It is less common in puerperal CVT with acute presentation of headache, seizures, focal deficits and altered sensorium. The incidence amongst Indian patients varies from 7.4-55%.[34,36,50] However, in cases with subacute onset a clinical picture stimulating idiopathic intracranial hypertension can be as high as 83-100%.[13,36]

Other uncommon clinical features are signs of meningeal irritation, cortical blindness, akinetic mutism, dystonia and associated venous thrombosis in the lower limbs or other parts of the body. Nagaraj et al.[50] grouped clinical features of CVT into four categories, depending on the topographic venous involvement.

Group-I: Meningo encephalitic type: headache, fever, seizures, altered sensorium, focal deficits, meningeal signs.

Group-II: Acute fulminant type :status epilepticus, coma.

Group-III: Pseudo tumour type : headache ,vomiting , pappiledema

Group-IV: Neuropsychiatric type : abnormal behaviour, with or without features of raised intracranial tension.

Cavernous sinus thrombosis

It is due to spread of infection from face, paranasal **s**inus or intracranial venous sinuses. It has a distinctive clinical picture where patient Presents with fever, chills, toxemia with proptosis, chemosis and painful **o**pthalmoplegia, initially unilateral but often becoming bilateral. Pappiledema and retinal hemorrhages indicate retinal vein thrombosis.

In a study conducted by ameri et al.[51] in a series of 110 patients with CVT, main signs and symptoms reported are the following

TABLE 2: CLINICAL FEATURES OF CEREBRAL VENOUS THROMBOSIS BY AMERI et al

SI NO	CLINICAL FEAUTURES	Ameri et al. (1992)
		N=110(%)
1	Head ache	83 (75%)
2	Papilloedema	54 (49%)
3	Motor or sensory deficit	38 (34%)
4	Seizures	41 (37%)
5	Altered sensorium	33 (30%)
6	Dysphasia	13 (12%)
7	Cranial nerve palsies	13 (12%)
8	Cerebellar incoordination	3 (3%)
9	Nystagmus	2 (2%)
10	Hearing loss	2 (2%)
11	Cortical signs	3 (3%)

In a recent Dutch European study, the most frequent symptoms and signs were:

1. Headache - 95%
2. Pappiledema - 49%
3. Seizures - 47%
4. Motor/sensory deficits - 34%
5. Changes in consciousness - 30%
6. Dysphasias - 12%
7. Cranial nerve palsies - 12%
8. Nystagmus - 2%
9. Deafness - 2%

TABLE3: CLINICAL FEATURES OF CEREBRAL VENOUS THROMBOSIS

Sl. No	Clinical Features	Nagaraja[50] (1980) N=138(%)	Bansal[40] (1983) N=135(%)	Srinivas[11] (1987) N=200(%)
1	Fever	62	16	11
2	Headache	48	24	57
3	Vomiting	36	24	-
4	Seizures (a) GTCS	29	50	35
	(b) Focal	17	22	23
	(c) Status epilepticus	-	-	14
5	Diplopia	1	-	-
6	Dysphasia	25	5	-
7	Nuchal rigidity	3	10	13
8	DVT	10	-	-
9	Altered sensorium	41	43	81
10	Pappiledema	35	16	14
11.	Ocular palsy	-	2	11
12.	Motor deficit (a) Monoplegia	5	9	-
	(b) Hemiplegia	53	37	-
	(c) Paraplegia	27	3	

COMPLICATIONS

1. Increasing venous congestion raises cerebrospinal pressure if collateral drainage is insufficient.
2. Parenchymal edema with infarction, hemorrhage complicates 50% case.
3. Seizures may persist requiring continued antiepileptics.
4. Pulmonary embolism is uncommon but carries a poor prognosis.[52]
5. Hypopituitarism may result from cavernous sinus thrombosis.
6. Dural sinus thrombosis has an etiological association with dural A-V fistulas.

INVESTIGATIONS

Computed tomography

CT scanning with and without injection of contrast material is usually the first neuroimaging examination carried out in patients with headache, focal deficits or seizures, particularly on an emergency basis. CT scanning is extremely useful for ruling out many CVT mimics. It occasionally detects lesions that can themselves cause CVT such as meningiomas, abscesses, sinusitis, and mastoiditis. CT scanning is also useful in showing brain or sinus changes suggestive of CVT.

Direct signs of cerebral venous thrombosis

Three abnormalities are considered direct signs of CVT:

1. The cord sign, 2.The dense triangle sign and 3. The empty delta sign.

Plain CT cord sign [53, 54]: Represents the thrombosed cortical vein. It appears as a linear hypodense streak in location of involved vein. It represents acute or new thrombus, thus seldom seen in chronic cases. The cord sign, visible on unenhanced CT scan, represents the spontaneous visualization of a thrombosed cortical vein; it is very rare and its diagnostic value is debated. The cord sign can also be seen in thrombosis of the internal cerebral veins and of the vein of galen.

Dense triangle sign[53, 54]: Triangular area of increased density along course of superior sagittal sinus and represents opacification by freshly coagulated blood. It is rare and usually seen in the first two weeks of the disease. It is a very early sign but an extremely rare one, being present in less than 2% of cases. The dense triangle is difficult to assess, particularly in other sinuses (lateral sinus and straight), which can be spontaneously hyper dense in normal children or in patients with hemo concentration.

The empty delta sign described by Buonanno and colleagues[54] appears after injection of contrast material. It reflects the contrast between the opacified collateral veins in the SSS wall and the non-opacification of the clot inside the sinus. It is the most common direct sign, present in approximately 35% of published cases.

The empty delta sign is absent, however, when (1) thrombosis does not affect the posterior third of the SSS or (2) CT scanning is performed either in the first 5 days or more than 2 months after onset of symptoms.

Indirect signs of cerebral venous thrombosis:

Indirect and nonspecific abnormalities are more common in CVT.

a. Cerebral edema. White matter hypo density without contrast enhancement suggestive of cerebral edema is present in up to 75% of cases. It can be diffuse· or localized and sometimes associated with a mass effect. This finding is usually associated with abnormalities suggestive of a venous infarct, but it can occasionally be the only sign of CVT.

b. Cerebral infarction not conforming to an arterial territory: Usually described by pathologists as hemorrhagic, venous infarcts on CT scan manifest as a spontaneous hyper density in 10% to 50% of cases.

c. Small ventricles: A common finding is the presence of small ventricles with swelling and sometimes diffuse low density suggestive of edema. Although reported in 20% to 50% of cases, this finding is not a useful sign because it is non-specific and is frequently difficult to differentiate from normal brain, particularly in the young.

d. Gyral and Tentorial enhancement: Tentorial enhancement is usually thought to suggest SS thrombosis but it is not rare in SSS thrombosis. It can be associated with dilated transcerebral medullary veins, indicating a major venous stasis, usually in relation to an extensive SSS thrombosis.

e. Non-hemorrhagic venous infarcts: They are almost as common. They are protean in appearance, taking the form of focal hypo density with gyral enhancement, areas of hypo density without enhancement or non hemorrhagic infarcts which can be unilateral or bilateral single or multiple. They are seen superficially in the hemispheres in SSS thrombosis and within the basal ganglia in deep venous system thrombosis.

f. **Bilateral signs**

g. **Subarachnoid hemorrhage**

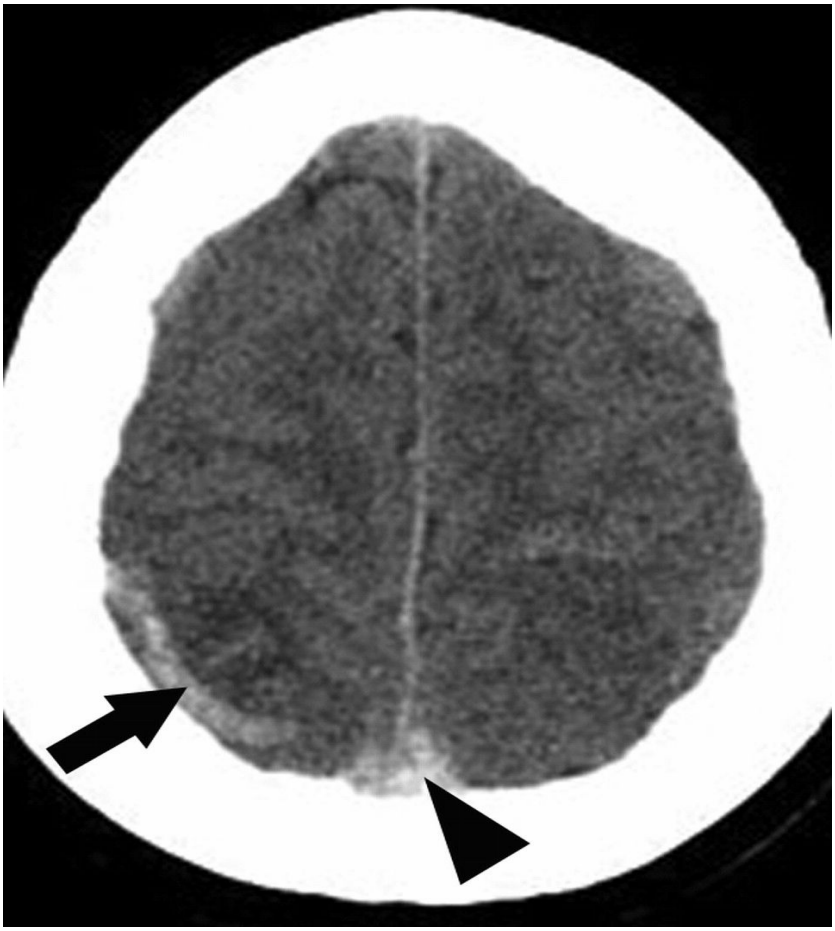
h. **Subdural hematoma**

i. Normal CT scan in CVT

In 10% to 20% of cases, CT scanning is normal in patients with proven CVT, more commonly (up to 50%) in patients presenting with isolated intracranial hypertension than (<10%) in those with focal signs[52,53]. The place of CT scanning in the diagnostic strategy of CVT is mainly to rule out other conditions, such as arterial stroke, abscess, tumors, and subarachnoid hemorrhage on an emergency basis. However, MRI or angiographic confirmation must be obtained.

Thrombosis most frequently affects (in order of decreasing frequency) the SSS, LS and cavernous sinus.

FIG-3 PLAIN CT-- DENSE TRIANGLE SIGN

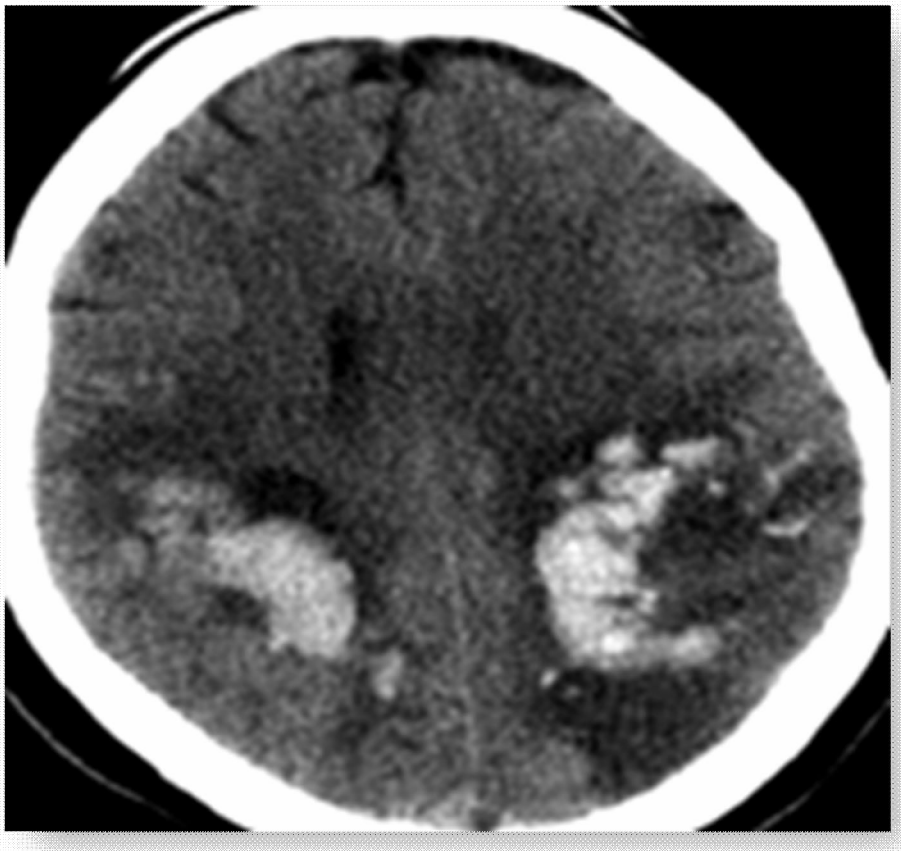


Axial unenhanced CT images show a “dense triangle” sign (arrowhead) in SSS and a cord sign (arrow), showing cortical vein thrombosis.

FIG 4-EMPTY DELTA SIGN

Contrast-enhanced CT image reveals low-attenuating thrombus (arrow) within the superior sagittal sinus, surrounded by a triangular area of enhancement.

Fig 5-BILATERAL HEMORRHAGIC INFARCT



Plain CT scan of head in axial plane shows hemorrhagic infarcts in bilateral parieto occipital lobes.

Magnetic resonance imaging (MRI) in venous thrombosis

MRI offers the following major advantages for the evaluation of possible CVT sensitivity to blood flow, ability to visualize the thrombus itself, and non invasiveness. A variety of MRI findings have been described.

1. **In acute phase-** Normal venous flow void is absent, and the occluded vessel appears isointense on T1weighted images and hypo intense on T2-weighted images. The diagnosis at that stage is often impossible on MRI alone. Angiography (or MRV) is required to demonstrate the absence of flow in the thrombosed vessel.

2. During 5-15 days the absence of flow void persists, but the thrombus becomes hyper intense, initially in T1 and then on T2 weighted images. In large vessels, these changes start in the periphery and proceed toward the center. They represent the aging of the thrombus with biochemical conversion of oxyhemoglobin to methemoglobin rather than extension of thrombosis.

This intermediate pattern signal on T1-and T2 weighted images is diagnostic of CVT and is by far the most common. It is usually found between 4-35 days after the onset of symptoms.

3. Late changes (approximately 2 to 4 weeks after onset)

It can reveal the beginning of vascular recanalization with the resumption of flow void in the previously thrombosed vessel.

However, at 6 months, more than two thirds of cases still show some heterogeneous localized signal abnormalities, which can persist for years and should not be mistaken for a recurrent acute CVT.

Besides visualizing the thrombus itself, MRI detects the parenchymal consequences. These include brain swelling with mass effect and cortical sulcal effacement; increased signal on T2 weighted images with isointense or hypo intense signal on T1 weighted images suggestive of edema and increased signal on both T1 and T2 weighted images, indicating a hemorrhagic component. These findings are nonspecific, but their diagnosis is easy because of the associated MRI signs of sinus thrombosis. The main difficulty is with isolated cortical vein thrombosis, which can be mistaken for a tumor unless angiography demonstrates typical stop sign with corkscrew collateral veins.

Diffusion-weighted imaging (DWI) seems to have several advantages. First, the clot can be directly visualized as high signal intensity in the affected sinus. Second, the diffusion pattern in the venous infarcts is different from that in arterial infarcts. The most common pattern is heterogeneous signal intensity with a normal or increased

apparent diffusion coefficient (ADC) corresponding mostly to vasogenic edema combined with some areas of cytotoxic edema.

Commonest venous channels involved in CVT in MRV are [57]:

1. Superior sagittal sinus thrombosis - 72%
2. Transverse sinus thrombosis - 70%
3. Straight sinus - 14%
4. Deep sinus - 8%
5. Cavernous sinus - 3%.
6. Cerebral veins - 38%
   - Superficial - 27%
   - Deep - 8%
   - Cerebellar veins - 3%

ANGIOGRAPHY

Intra-arterial angiography

Angiography had been the key procedure in the diagnosis of CVT for many years and remains the method of reference in some difficult cases. It requires four-vessel angiography conventional or digitized intra-arterial. The partial or complete lack of filling of veins or sinuses is the best angiographic sign of CVT. It is easily recognized when it affects the

posterior or whole SSS, both lateral sinuses and the deep venous system. The lack of filling may be more difficult to interpret in other locations, such as the anterior third of the SSS or the left lateral sinus. However, in such cases, signs are lacking and MRI is required to differentiate between thrombosis and hypoplasia.

Another angiographic finding is delayed emptying, collateral venous pathways, found in about 50% of cases, almost invariably indicate SSS thrombosis. Dilated and tortuous cortical collateral veins with a corkscrew appearance are much more common than transcerebral or intra dural collateral vessels.

Magnetic resonance venography

The current practice is to use noninvasive magnetic resonance venography (MRV) instead of intra-arterial angiography for the diagnosis of CVT. A four vessel angiography with venous phase films taken at intervals of 5-12 seconds of contrast injection. Frontal, lateral, oblique views are taken in order to visualize the entire venous system. Delayed venous films may be necessary to allow for slow venous filling in patients with raised intracranial tension. Angiography becomes necessary if thrombolytic therapy is considered.

Findings

1. Failure to demonstrate all or part of sinus / vein or localized irregularity within its wall.
2. Non filling of cortical veins.
3. Focal or diffuse slowing of circulation and stagnation of coagulated blood in capillary or venous phase.
4. Corkscrew vessels that do not reach cortical surface due to dilatation of anastomotic vein over cortex.
5. Parenchymal changes include:
    - Focal or diffuse brain swelling with sulcal effacement
    - Intracranial hemorrhage
    - Ventriculomegaly due to decreased cerebrospinal fluid, a consequence of increased resistance of CSF resorption by the arachnoid granulations.

Helical CT venography

Helical cerebral CT venography has been developed to study the venous circulation. Filling defects, sinus wall enhancement, abnormal collateral venous drainage and tentorial enhancement are common abnormalities. It has been suggested that CT venograms are easier to interpret and have fewer artifacts than MRV. CT venography is particularly interesting in the acute setting because it can be easily

performed immediately after contrast-enhanced CT, which is usually the first procedure performed in emergencies.

IV. Radionuclide Scanning: Dynamic radionuclide scanning has been utilized for visualization of venous dural sinuses, but lacks sensitivity and specificity.

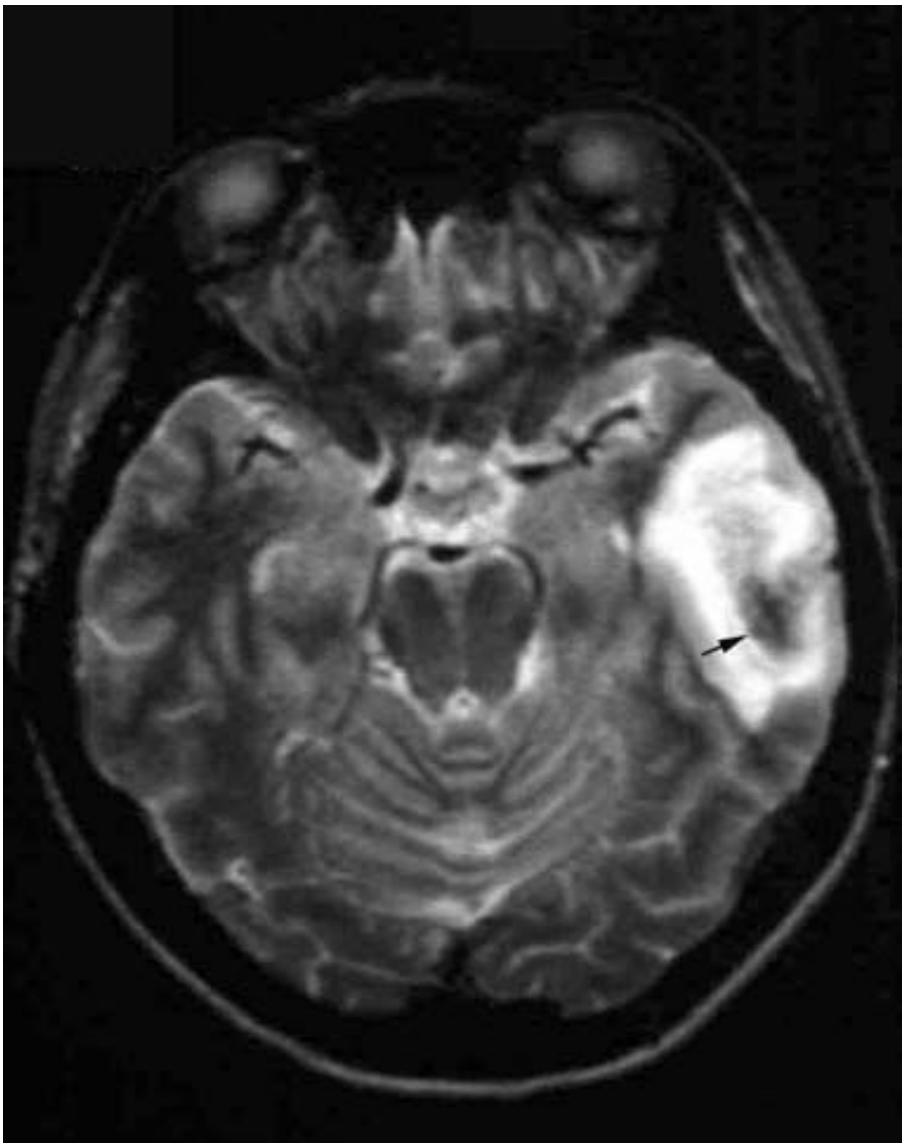
V. Other Investigations

1. CSF examination: It is of diagnostic use .It is performed in suspected benign intracranial hypertension, to rule out CVT associated with infections, carcinomatous meningitis etc .It may reveal non specific changes like raised proteins ,cells and pressure.

2. Appropriate tests towards etiogenesis like:

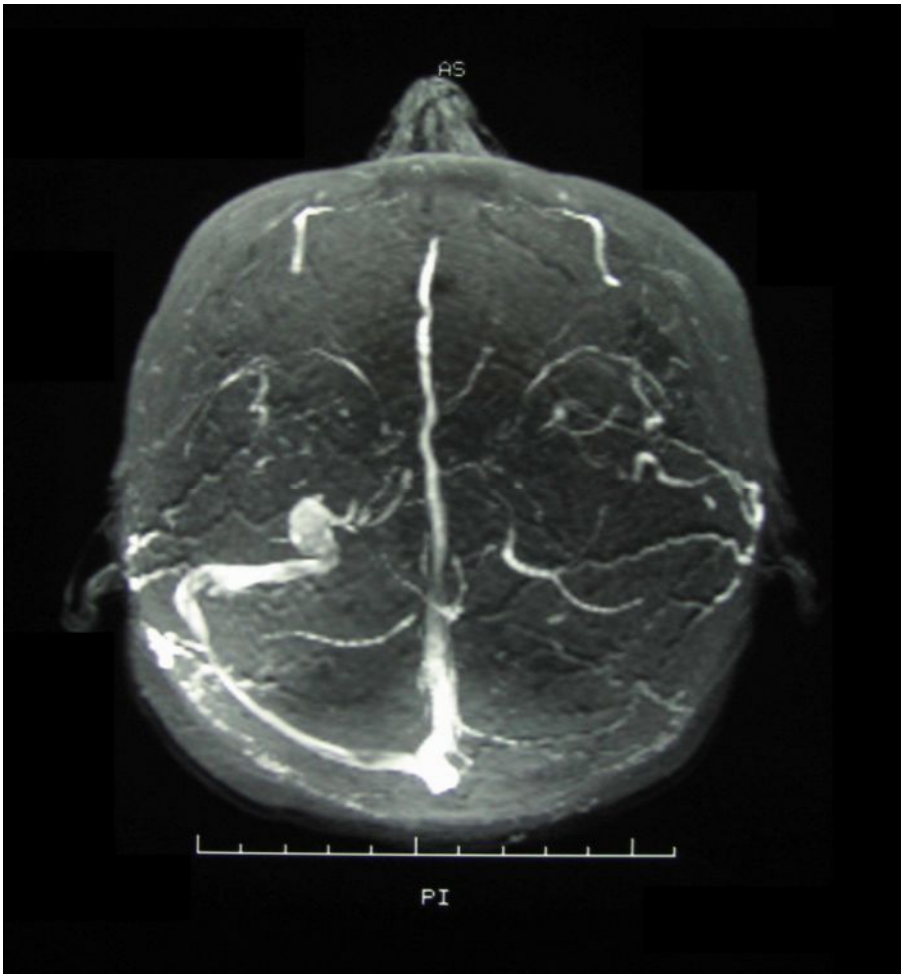
- Complete blood count may reveal anemia, polycythemia, leucocytosis, and thrombocytopenia.
- Lupus anticoagulant and anti cardiolipin antibodies.
- Protein C and S,Antithrombin III, Factor V Leiden mutation.
- Homocysteine.
- ESR, ANA ACL AB tests in connective tissue disorder.
- ANCA in Wegener's granulomatosis
- Urine for protein
- Liver function test
- D-dimer[58]
- ECG, ECHO, EEG

FIG 6-LEFT TEMPORAL HAEMORRHAGIC INFARCT



Axial T2-Weighted Image Reveals Left Temporal Lobe T2 Hyperintensity Consistent with Infarction and a Central Focus of T2 Hypointensity (Arrow) Due to Hemorrhagic Venous Infarction.

FIG-7 MAGNETIC RESONANCE VENOGRAM



Magnetic resonance venogram demonstrating occlusion of the left sigmoid and transverse sinuses.

TREATMENT

General Measures:

Include routine measures for care of comatose patient and includes:

1. Maintenance of airway patency.
2. Maintenance of fluid and electrolyte balance.
3. Prevention of infections and gastric ulcers.

Symptomatic Treatment:

- **Seizures**: Antiepileptics are obviously indicated in patients with clinical seizures.

2. Cerebral edema: Cytotoxic edema results due to alteration in biochemical mechanism at cellular level. It is followed by vasogenic edema in 24-48 hours Measures to control cerebral edema and raised intra cranial tension are:

- Head end elevation to 30 degrees to promote venous drainage.
- Mannitol (20%)-in dose of 0.25-1g/kg i.v over period of 10minutes
- Dexamethasone given in a dose of 4mg every 6 hours. It primarily reduces vasogenic edema.

- In a patient with large venous infarcts with mass effect raises the intracranial pressure considerably and if vision continues to deteriorate, in such cases besides starting mannitol, oral glycerol or acetazolamide, intravenous steroids repeated lumbar puncture and surgical decompression like lumboperitoneal shunt have been suggested[58].

Etiologic treatment

Whenever possible, etiological cause of CVT should be treated. It includes wide spectrum antibiotics for septic CVT and surgical debridement for primary site of infection. Malignancies, collagen vascular disease and hematologic disorders need specific treatment.

Antithrombotic treatment

Although heparin was first recommended 6 years ago, some were skeptical about its use owing to the fear of further hemorrhage into hemorrhagic infarcts. However there is ample evidence favoring the use of heparin for patents with CVT even if there are hemorrhagic infarcts in CT or MRI.[59,60,61] Anticoagulants are used to prevent propagation of the clot to more extensive areas of cerebral venous system and thereby prevent progression of infarction. Neonatal CVT is the only situation in

which heparin has not been shown to improve the outcome and most authors do not recommend its use in this situation (Nagaraja[61], Sarma 2002). The antithrombotic treatment modalities include heparin, thrombolysis and oral anticoagulants.

A. Heparin: Increases the action of antithrombin III, leading to inactivation of coagulation factors thrombin, factor Xa and IXa. DOSE: Most studies have used higher doses of heparin. The dose of heparin used should result in aPTT twice of the baseline. Both low molecular and unfractionated heparin are equally efficacious. Heparin is given parenterally for 2 weeks followed by oral anticoagulants for 3-6 months. Unfractionated heparin can be given either by subcutaneous or continuous IV infusion.

B. Warfarin: Acts by interfering with the action of vitamin K, a cofactor essential for converting precursor proteins into factors 2,7,9,10.

It is used for 3-6 months following parenteral heparin. Dose is 5mg/day; dose adjusted by monitoring prothrombin time and INR.The dose is adjusted to obtain an INR between 2 and 3.

Oral anticoagulants are continued for 6 months when there is a known acute cause of CVT like postpartum state, minor head trauma or

local infection. In contrast, prolonged treatment is recommended when there is continuing risk of thrombosis as with inherited thrombophilia, prothrombotic states associated with malignancies and inflammatory disorders like behcet's disease or systemic lupus erythematosis.

Thrombolytic therapy

Intravenous urokinase was used for CVT, first by Vines and Dand in 1971 and ten years later by Di rocco and colleagues; direct endovascular thrombolytic agents like urokinase, RT-PA are being administered into dural sinuses for recanalisation. Two series totaling 21 patients have been reported in literature of its use in dural sinus thrombosis [62, 63] administered through jugular catheter. In most cases outcome was good. Nevertheless both carry a risk of hemorrhage being advocated especially in patients in whom the medical therapy has failed.

Surgery

Surgical decompressive craniotomy, thrombectomy and evacuation of intracerebral hematoma need further evaluation before it is recommended as a safe procedure. Out comes are usually poor.

OUTCOME

Short term outcome

The three main causes of death in CVT are:

1) Brain lesion itself, particularly when a large hemorrhagic infarct is present
2) Intercurrent complications like sepsis, uncontrolled seizures and pulmonary Embolism
3) Underlying conditions like carcinoma, septicemia, leukemia and paroxysmal nocturnal hemoglobinuria.

Factors classically considered to suggest a bad prognosis are as follows:

- *Rate of evolution of thrombus*
- *Age of the patient*
- *Infection as a cause*
- *Focal symptoms and coma*
- *presence of hemorrhagic infarct*
- *Empty delta sign on CT scan*
- *Topography of cerebral veins involved.*

Among all underlying conditions, the postpartum state is a favorable one with a survival of 90% in most series.

Long term outcome

If the patent with CVT survives, the prognosis for recovery of function is much better than for patients with arterial thrombosis. Residual epilepsy has been reported in 10-30% of patents who had seizures during the acute stage of CVT. Recurrence of venous thrombosis at another site can occur in patents with prothrombotic states, but is generally uncommon with long term anticoagulation.

One hundred of 138 cases of cerebral venous thrombosis related to pregnancy and the puerperium recovered completely in the series of Bansal et al.[64], Srinivasan et al[65] observed that of 135 patients (129 venous thrombosis and 6 Arterial thrombosis) in pregnancy and puerperium[80] recovered without significant neurological deficit. Fifty of these cases followed-up for two years were doing well. Ten patients had residual focal neurologic deficit without disability; 10 were active but had recurrent though infrequent seizures. Stanisfile[66] on the basis of 16 cases from literature reported a mortality rate of 56%, Warle kendall[67] gave a figure of 30%.

MATERIALS AND METHODS

METHODOLOGY

Forty (40) patients admitted to the Medical Emergency Ward, clinically suspected of CVT, during the period of September 2010 to September 2011 were subjected to neuro imaging techniques, fulfilling the study criteria were recruited by simple random sampling and data collected was analyzed by correlation studies.

Study Design

Prospective, clinical study with CT or MRI

Study center

Institute of Internal medicine at Government General Hospital (GGH), Chennai

Selection of patients

Inclusion Criteria:

40 consecutive patients presenting with history suggestive of cerebral venous thrombosis and CT scan direct and indirect signs confirmed by imaging of brain (MRI & MRV).

A. Direct Signs

a. Hyperdense sinus on plain CT

b. Cord sign on plain CT

c. Empty delta sign on contrast enhanced CT

d. Dense triangle sign on plain CT

B. Indirect Signs

a. Cerebral edema

b. Cerebral infarction not conforming to an arterial territory

c. Small ventricles

d. Bilateral signs

e. Gyral enhancement

f. Tentorial enhancement.

Exclusion Criteria:

1. CT scan inconclusive of CVT
2. Hypertensive hemorrhage
3. Arterial stroke
4. Metabolic encephalopathy
5. Space occupying lesions.

Data was collected by using pre-tested proforma meeting the objectives of the study. Purpose of the study was carefully explained to the patients and informed consent was taken. All patients were interviewed. Detailed history was taken with respect to epidemiological, clinical features, radiological features, with special emphasis on suspected precipitating or predisposing factors such as puerperium, fever, sepsis, anemia, abortions and oral contraception.

Detailed examination of patients was carried out including general physical examination for any evidence of anemia, dehydration, sepsis, deep vein thrombosis of leg and detailed neurological assessment with other systems were done to look for any evidence of etiologies.

OBSERVATIONS AND RESULTS

OBSERVATION AND RESULTS

A total of 40 cases of cerebral venous thrombosis were evaluated in the Present study.

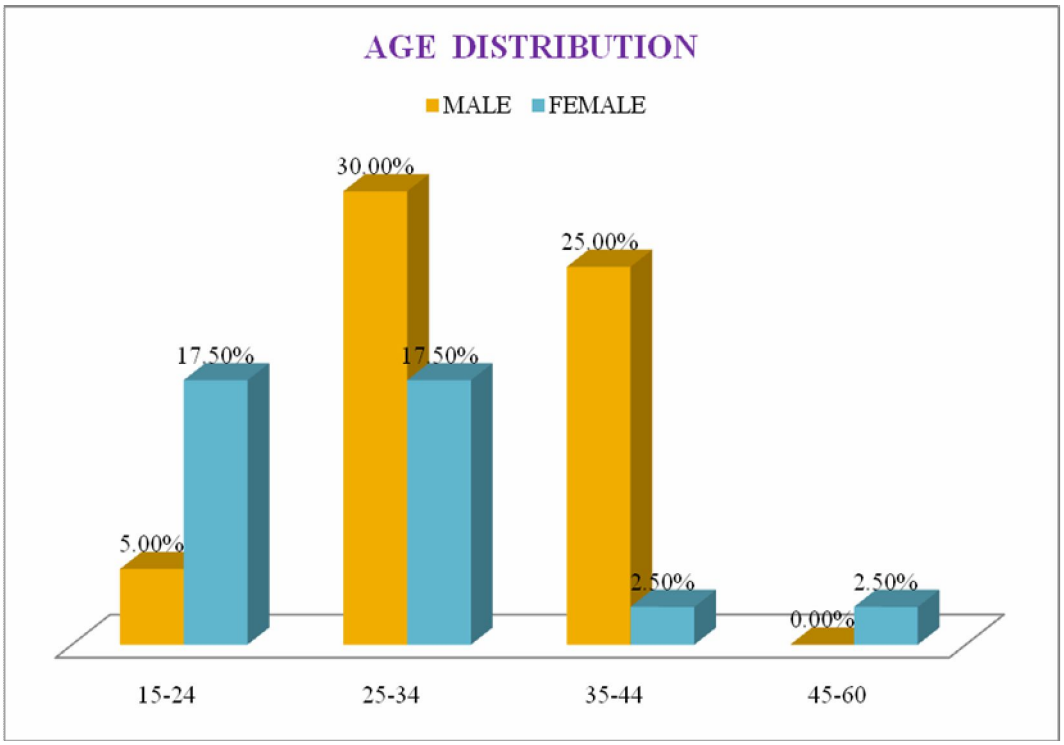
1. Age distribution

TABLE 4- AGE INCIDENCE

Sl.No.	Age in years	Number of cases	Percentage
1	15-24	8	20
2	25-34	22	55
3	35-44	8	20
4	45-60	2	5

The mean total cohort age is 30.05.the mean age for puerperal age group is 25.62.

FIGURE 8: AGE INCIDENCE



The mean age of the patients in the present study was 30.05%. Majority of them were in the age group 15-35 years contributing to 75%. The youngest age being 20 and highest is 55 years.

2. Sex distribution

TABLE 5- SEX INCIDENCE

Sl. No.	Sex	Total number of cases (n=40)	Percentage
1	Females	16	40
2	Males	24	60

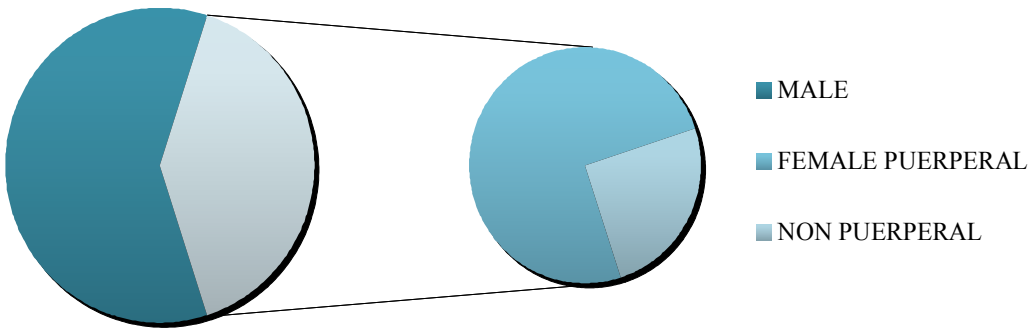
3. Types of CVT

TABLE 6- TYPES OF CVT

Category	Total number of female cases(n=16)	Percentage in female cases	Percentage in total study.(n=40)
Puerperal	14	87.5	14 (35%)
Non puerperal	2	12.5	26(65%)

There were 24 men (60%) and 16 women (40%) in the study. Out of 16 female patients, 14 (87.5%) were puerperal and 2 (12.5%) were non puerperal CVT.

FIG 9-SEX INCIDENCE



Here male: female ratio is 1.5:1. In the present study, out of 40, 26 (65%) patients belong to non puerperal group and 14 (35%) belong to puerperal group.

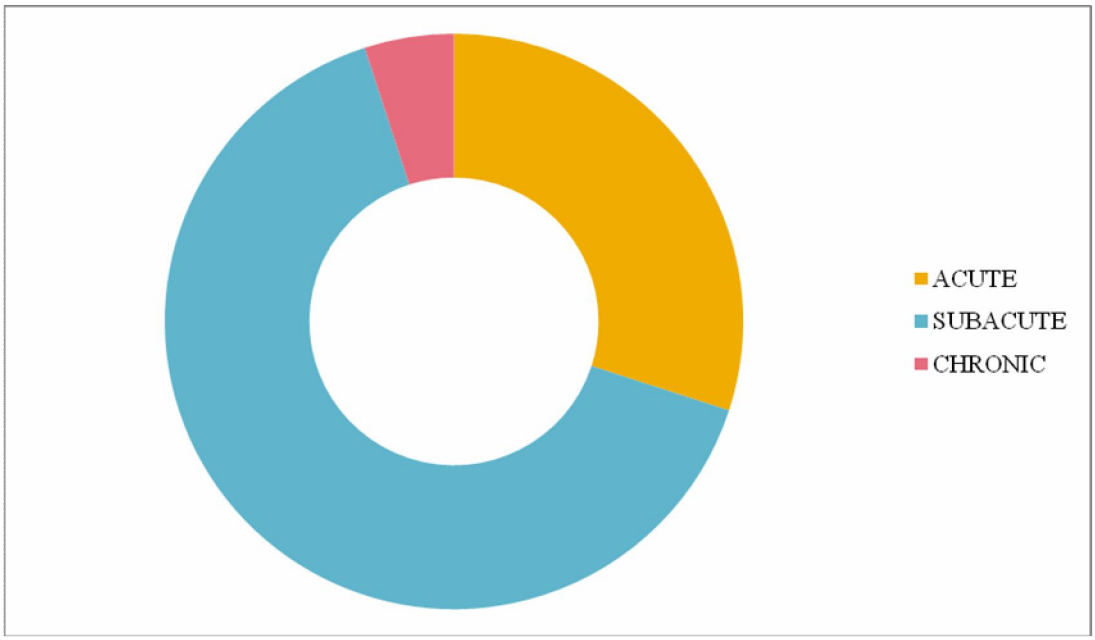
4. Mode of onset

Those who presented within 48 hours were considered to have acute onset,with onset longer than 48 hours but less than 1 month were considered subacute, and with onset more than 1 month as chronic (bousser et al., 1985)

TABLE- 7: MODE OF ONSET

Mode of onset	Number of cases	Percentage
Acute	12	30
Sub acute	26	65
Chronic	2	5

FIG 10-MODE OF ONSET



In the present study, 26 cases (65%) of CVT had sub acute presentation, followed by 12 cases (30%) with acute presentation and 2 (5%) cases had chronic presentation.

5. Level of consciousness at the time of presentation

TABLE 8- LEVEL OF CONSCIOUSNESS

Level of consciousness	No. of patients (n=40)	Percentage
Conscious	14	35
Drowsy	12	30
Stuperous	8	20
Comatose	6	15

In the present study, patients presented with full consciousness include 14 cases contributing 35%. 12 cases were drowsy with 30% incidence.8 (20%) were stuperous and 6 (15%) came with coma.Death is common in comatose patients.

CLINICAL SIGNS AND SYMPTOMS

6. Initial onset of symptoms

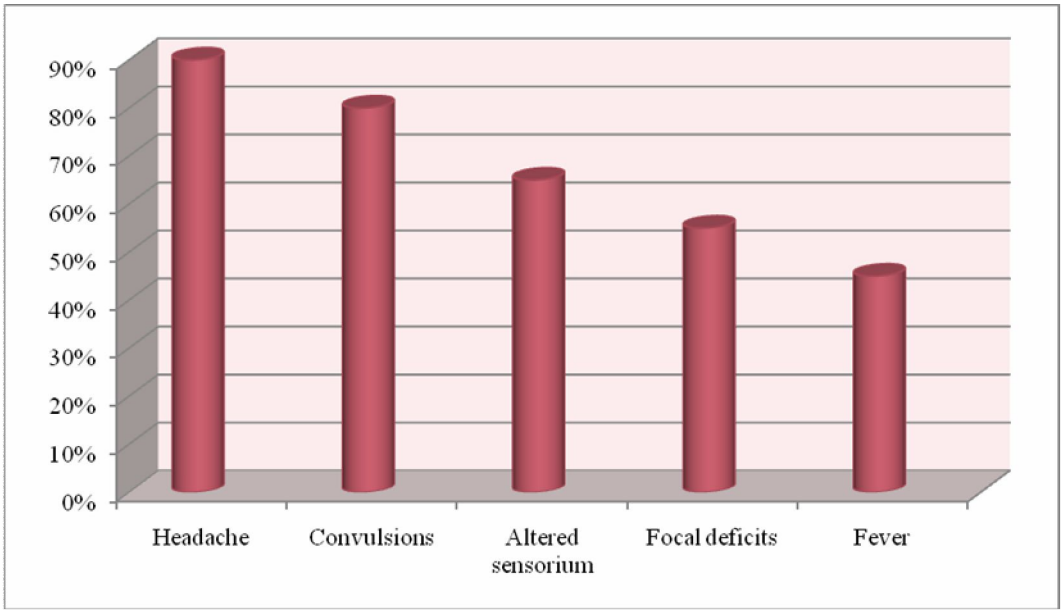
Patients with initial symptom of presentation was taken into consideration.

TABLE 9- INITIAL SYMPTOMS

Symptoms	Number of cases	Percentage
Headache	36	90
Convulsions	32	80
Altered sensorium	26	65
Focal deficits	22	55
Fever	18	45

In the present study, most common symptom was headache contributing to 87.5% (36 cases) followed by convulsions in 80% (32 cases).Two of them had ear discharge and another with diarrhea.

FIGURE 11: INITIAL PRESENTATION



In our study, 65% presented with altered sensorium, 55% with focal deficits, 45% with fever.

7. Neurological signs

TABLE 10- NEUROLOGICAL SIGNS

Signs		Number of Cases	Percentage
Focal deficit		22	55
Fundus	Disk bulge	12	30
	Pappiledema	8	20
Cranial nerves		14	35

In the present study, hemiplegia was present in 55%, cranial nerve involvement in 35% and Pappiledema in 20% of patients.

INVESTIGATIONS

8. Hemoglobin percentage

TABLE- 11: HEMOGLOBIN PERCENTAGE

Hb%	Number of cases	Patients alive	Patients dead
2-5	1	1	-
5.1-8	6	5	1
8.1-10	10	8	2
> 10	23	18	5

In the present study, out of 40, 17 patients were anemic, accounting for 42.5%. The maximum number of deaths appears to be more with HB% > 10 g%, and the percentage of mortality was higher in non anemic group.All patients who died with mild and moderate anaemia were in the puerperal group.

9. CSF Analysis (done in 18 patients)

TABLE 12: CSF ANALYSIS

CSF changes	Number of Patients
Normal	**8**
Protein rise	**4**
Pleocytosis	**6**
Xanthochromia	**2**

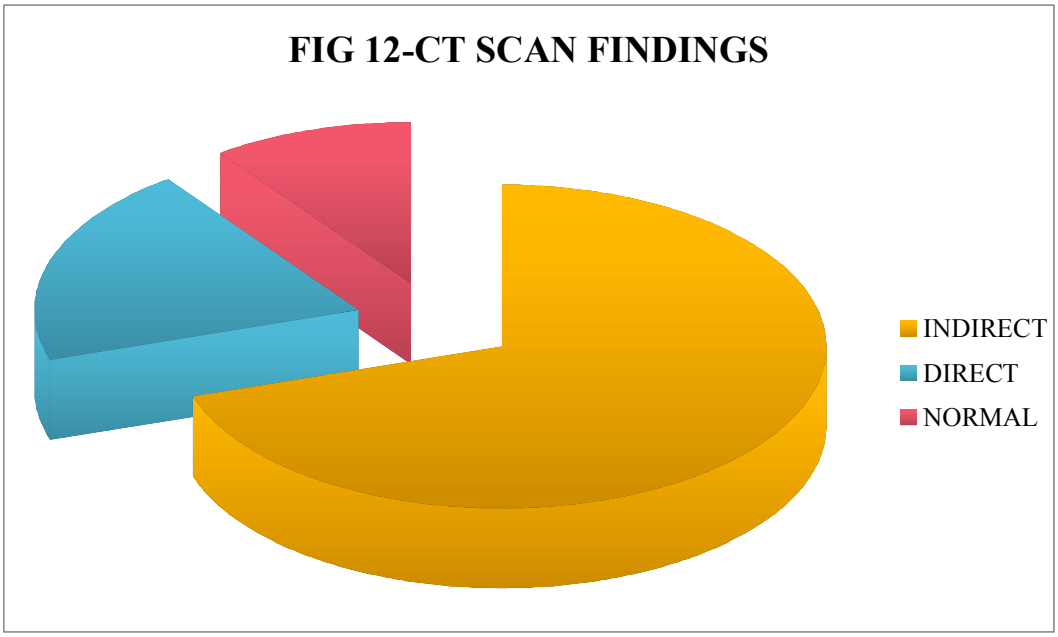
Eighteen (18) patients were subjected to CSF analysis whenever there was suspicion of meningitis of which 8 were normal and abnormality seen in rest 10 patients with pleocytosis being the maximum. Two patients had TB meningitis.

10. CT Scan findings

TABLE 13: CT SCAN FINDINGS

Signs	Number of Cases	Percentage
Cord sign	4	10
Empty delta sign	3	7.5
Dense triangle sign	1	2.5
Mass effect	5	12.5
Midline shift	5	12.5
Bilateral infarct	3	7.5
Edema	8	20
Hemorrhagic infarct	22	55
Non-haemorrhagic infarct	2	5
Normal	4	10

FIG 12-CT SCAN FINDINGS



In the present study, Haemorrhagic infarct comprises 22 cases with 55% followed by edema in 20%.Out of 40 patients 10% had normal CT picture. Indirect signs (70%) were more common than direct signs (20%).

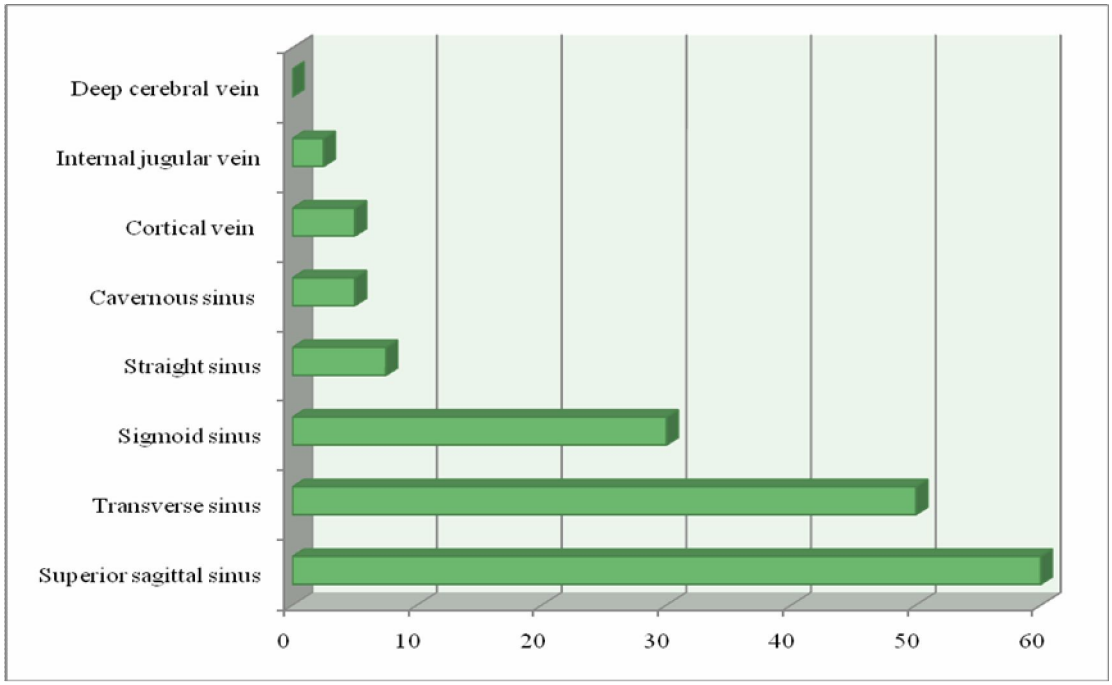
11. Sinuses involved in MRI+ MRV.

All 40 cases, including 10 patients with normal CT scan findings underwent MRI scan.

TABLE 14: SINUS INVOLVED IN MRI+MRV

Sinus involved	Number of Cases	Percentage
Superior sagittal sinus	24	60
Transverse sinus	20	50
Straight sinus	3	7.5
Sigmoid sinus	12	30
Cavernous sinus	2	5
Cortical vein	2	5
Internal jugular vein	1	2.5
Deep cerebral vein	-	-

FIGURE 13: SINUS INVOLVED IN MRI + MRV



In the present study, the most common sinus involved was superior sagittal Sinus in 24 patients accounting to 60% followed by transverse sinus in 20 Patients 50%.

12. Predisposing or etiology factor

Predisposing/ etiology factor were identified in 25 (62.5%) of patients. In 15 (37.5%) risk factor could not be identified. Rheumatological evaluation was done only in 10 patients because of cost restraints. Also we found 11 patients had the habit of using smoke and alcohol, 4 were only smokers, 1 was an alcoholic and 6 were pan chewers. All these lead to hypercoagulable state.

Infections-7

HIV-2

TBM-2

CSOM-2

Diarrhoea-1

Non Infectious

Hyperhomocysteinemia-17

APLAS-2

Protein C/S deficiency-1

Puerperal -14

PIH-2

13. Clinical outcome

TABLE 15: TOTAL MORTALITY

Category	No of patients		Mortality	Percentage
Male	24		5	12.5
Female	Puerperal	14	3	7.5
	Non-puerperal	2	-	-

In this study of 40 patients, 32 were alive attributing to 80% and 8 died comprising 20%. 12.5% died in non-puerperal group and 7.5% died in the puerperal group.

DISCUSSION

DISCUSSION

The first description of CVT was by Ribes[11] in 1825 as a postmortem report in French literature. Most of the initial reports are from autopsy findings and it was found to be extremely low. Ehlers[14] found only 16 SSS thrombosis in a 12500 autopsy series. Kalbag and Wolf [68]indicated that CVT was the principal cause of death in only 1 per million (21.7 persons per year) in England and Whales between 1952 and 1961.At Nimhans , Bangalore, of the 1760 brains examined 75 were of primary CVT (Nagaraja et al[34]). But recent studies like Bansal et al[40], Rao et al[46] suggests that the true incidence is much higher than that thought from the initial autopsy series.

Cerebral venous thrombosis though not rare, is often undiagnosed as it presents with a wide array of symptoms .It mimics practically all neurological conditions. It affects all age groups and has unpredictable outcome. This condition is common in the Indian subcontinent, responsible for 10-15% of young strokes.

It has been reported more commonly in women. Studies by various Indian authors (Agarwal1978, Srinivasan1983, Banerjee1973, Nagarajan 1988) have highlighted higher prevalence in pregnancy and puerperium.

Though our study of 40 patients does not give the real incidence of the disease the fact that they were seen in 1 year at Government general hospital, Chennai suggests that CVT is not uncommon.

1. Mean Age of onset

TAB- 16: MEAN AGE OF ONSET

Authors name	Mean age of onset (years)
Daif et al.[21]	27.8
Nagaraj et al.[36]	24.2
Strolz e et al.[26]	32.8
Bousser et al[3]	40.8
Deschiens et al[69]	36.2
Zhang et al [71]	31
De brujjin et al[53]	37
Terazzi et al[70]	44.8
Present study	30.05

Comparing the age group involved, 15-35 years was the commonest age group involved in various series. The present study also showed similar findings with 75% in the same age Group, with mean age of onset 30.05 years which is comparable with Zhang et al., Strolz e et al. It is found that the lower mean age of onset (25.62) is correlated with puerperal age group

2. Age Distribution

TABLE-17: AGE DISTRIBUTION

Author	Age in years			
	15-24	25-34	35-44	45-60
Carol et al[22]	25%	50%	25%	-
Bansal et al[40]	15.9%	65.2%	18.8%	-
Bousser et al[3]	5.3%	26.3%	18.4%	50%
Deschiens et al[69]	5%	27.5%	37.5%	30%
Present study	20%	55%	20%	5%

The age distribution in our study is similar to most other studies. About 75% of the patients in our series had onset between 15 and 34 years of age. 20%of the patients had onset between 15and 24. Most of these cases were puerperal.

2. Sex distribution

TABLE -18 : SEX DISTRIBUTION

Author	males	females
Bousser et al[6]	21 (55.3%)	17 (44.7%)
Daif et al.[21]	20 (50%)	20 (50%)
Deschiens et al[69]	10 (25%)	30 (75%)
Zhang et al[71]	9 (39.1%)	14 (60.9%)
De brujjin et al[25]	9(16.3%)	50 (84.7%)
Terazzi et al[70]	10 (30.9%)	38 (70.1%)
Present study	24 (60%)	16 (40%)

In our study sex distribution is almost similar to Bousser et al, Daif aet al. Most of the other studies reported as increased female incidence. Here ratio of male: female is 1.5:1

3. Types of CVT patients

In the present study, out of 40, 26 (65%) patients belong to non puerperal group and 14 (35%) belong to puerperal group.

Only 1 (2.6%) of the 38 patients of Bousser et al[6] was puerperal while in Deschiens et al study only 8(20%) of patients were puerperal. Only 1 (2.5%) of Daif et al. was post partum.4 (21.5%) of 19 patients of Zuber et al was related to pregnancy. Similar to these most of our cases were non-puerperal. This is because there is no obstetric department in our hospital and cases have to be referred from outside.

This is in contrast to previous Indian studies. Nagaraj et al. (1987) had found that 200 out of 230 cases (86%) of CVT, seen over eight years, were puerperal in nature. In a study by Srinivasan et al 25% maternal mortality is in CVT patients and complicate 4.5% of obstetric admissions.

SYMPTOMS AND CLINICAL FINDINGS

4. Mode of onset

In the present study, 26 cases (65%) of CVT had sub acute presentation, followed by 12 cases (30%) with acute presentation and 2 (5%) cases had chronic presentation.It is correlated with Nagaraj et al. (1987), Strolz e et al. (2005).

5. Level of consciousness

Comatose, drowsy and stuperous patients were calculated as patients with altered sensorium.

TABLE- 19: LEVEL OF CONSCIOUSNESS

Authors	Number of Cases studied	Percentage with Altered Sensorium
Bousser et al.(1985)[3]	38	26
Nagaraj et al. (1987)[37]	200	81
Ameri et al. (1992)[72]	110	30
Nagaraj et al. (1999)[61]	73	57.53
Neki s et al. (2003)[73]	14	56
Strolz e et al. (2005)[26]	79	36.7
Present study	40	65

In the present study, 65% of patients had altered level of consciousness which is comparable with Nagaraj et al.

5. Headache

Headache in the absence of intracranial hypertension, subarachnoid haemorrhage, or meningitis. Such cases are essentially associated with lateral sinus thrombosis , which should not be mistaken for lateral sinus hypoplasia.

Headache was the most common symptom in the present study accounting for87.5% of patients. The present study was comparable with most other studies like Neki s et al. with 85.5%, Daif et al. with 82% and mehta et al.[74] with 77.8%.

6. Seizures

The manifestations that indicate the cerebral cortical involvement are convulsions and paralysis, at times seizures are heralding symptoms and should arouse the suspicion of diagnosis.

TABLE- 20: SEIZURES

Authors	Number of Cases studied	Percentage with Seizures
Bousser et al. (1985)[3]	38	29
Nagaraja et al. (1992)[50]	200	70
Kumar s et al. (2003)[75]	85	67
Mehta sr et al. (2003)[74]	43	26.6
Srinivasan et al. (2005)[44]	135	68
Present study	40	80

In the present study, 80% of cases had seizures which are comparable with Nagaraja et al, Kumar s et al. In contrast, other studies showed less compared to present study.

Nagaraja et al found generalized seizures in 35 % and focal seizures in 23%, and 14% of them had status epilepticus of 200 patients. In our study 20% had focal seizures, 45 had generalized 10%with status and focal seizures with secondary generalisation in 25%.

7. Focal deficits

In the present study, 55% of patients had focal deficits. Among them 50% had hemiparesis and 5% had monoplegia. 35% had associated cranial nerve palsies, most commonly affected being the VI nerve. Strolz e et al. (2005) had found that out of 79 cases, 56.9% cases had hemiparesis. The present study was comparable with strolz e et al.

8. Papilledema

TABLE -21: PAPILLEDEMA

Authors	Number of cases	Percentage with papilledema
Nagaraja et al	200	15
Bousser et al.	38	45
Kumar s et al.	85	32
Neki s et al.	14	80
Mehta et al.	43	77.8
Daif et al	40	66.7
Present study	40	50

In the present study, 50% of patients had papilledema. Similar observations were noted with Bousser et al and Daif et al.

INVESTIGATIONS

9. Anemia

Anemia has often been noted in 17 (42.5%) of patients in the present study. The total number of deaths appears to be more with HB% > 10 g%, and the percentage of mortality was higher in non anemic group. This is because majority of the mortality fall in the non puerperal group.

The investigative procedures like leucocytes count, blood sugar, ESR did not contribute to the diagnosis and were non-specific. Except five cases where the blood Urea levels were high where patients had developed pre-renal failure due to fluid restriction.

10. CSF Analysis

In the present study, CSF analysis done in 18 patients which showed non-specific changes like Pleocytosis (> 5 cells/ mm^3 in 6 patients), raised proteins (> 45 mg/dl in 4 patients) and xanthochromia (2 patients).In large number of patients, CSF did not contribute to the diagnosis of CVT except the 2 who had TB meningitis.[76]

11. CT scan findings

As discussed earlier CT scan plain and contrast is the first neuro imaging technique to carry out when CVT is clinically suspected. It is useful to diagnose CVT and to rule out tumors, infarcts and hemorrhages.

The features primarily due to thrombosis of veins or sinuses are called direct signs and those due to secondary effects on brain parenchyma are referred as indirect signs.

TABLE- 22: CT SCAN FINDINGS

Signs	Rao et al (31)	Nagaraja et al (68)	Present study (40)
Cord sign	6.4	21.9	10
Empty delta sign	35.5	32	7.5
Dense triangle sign	-	-	2.5
Mass effect	-	8.1	12.5
Midline shift	-	3.1	12.5
Bilateral infarct	-	-	7.5
Edema	21.4	19.8	20
Hemorrhagic infarct	19.7	40.9	55
Non-haemorrhagic infarct	9.7	51.6	5
Normal	9.7	10.9	10

CT scans are normal in 10-20% of the patients in Bousser et al, Nagaraja et al, Rao et al. In our study 10% had normal CT pictures. 20% of 40 cases had direct evidence. In our study indirect signs (70%) were more common than direct signs (20%). The most common finding is hemorrhagic infarction present in 55% of cases. Similar observations noted with various studies like Nagaraj et al. and dixit et al. with 40.9% and 48.4% respectively.

12. Sinuses involved in MRI

MRI with MRA was done in all 40 patients. It was abnormal in all the patients.

TABLE- 23: SINUSES INVOLVED IN MRI

Sinuses	Ameri et al.	Daif et al.	Strolz e et al.	Present study
Superior sagittal Sinus	72%	85%	72.2%	60
Transverse sinus	70%	2.5%	38%	50
Straight sinus	16%	7%	7.6%	7.5
Sigmoid sinus	-	32%	20.3%	30
Cavernous sinus	-	-	-	5
Cortical vein	-	-	-	5
Internal jugular vein	-	-	7.6%	2.5
Deep cerebral vein	8%	10%	6.3%	-

In the present study, the superior sagittal sinus is most commonly involved[77] accounting for 60% followed by transverse sinus with 50%, comparable with other studies like strolz e et al. (72.2%) and ameri et al. (72%).

13. Predisposing or Etiology factor

Predisposing/ etiology factor were identified in 25 (62.5%) of patients. In 15 (37.5%) risk factor could not be identified. Infections were identified as risk factors in 7 (17.5%) cases. HIV was associated with 2 patients and they had chronic onset of illness, TBM in 2 , diarrhea in1,CSOM in 2 patients. Among non infectious puerperium was the common predisposing factor, in which 2 patient had PIH. 2 male patients had APLA (5%)[79] and combined Protein C/S deficiency in one patient[78]. 11 patients were chronic smoker and alcoholic, 4 were only smokers, 1 was an alcoholic and 6 were pan users. Out of this 17 patients had hyper homocysteinemia. Less marked elevations are proven risk factors for cardiovascular and venous thrombotic events[80]. Normal levels 5-15 μ mol/L.

14. Treatment

The role of Heparin in the management of CVT has been a matter of controversy. It's effectiveness, even in hemorrhagic venous infarcts has been extensively studied and recommended by various authors Einhaupl 1991[82], Cepri 1998[83], Nagarajan 1998[84]. Though most series have used high dose heparin as a continuous infusion in management of

CVT ,in the present study all 40 cases were treated with unfractionated Heparin 5000 i.v 8th hourly. All patients received oral anticoagulant for 3months. No complications were detected as a result of treatment regimen employed in the present study and proved to be effective.

15. Mortality

TABLE- 24: MORTALITY

Author	Number of cases	Percentage of Mortality
Bansal et al.	138	27.5
Srinivasan et al.	135	25.9
Nagaraj et al	200	21.7
Storlz e et al.	79	15
Present study	40	20

In the present study, the mortality is 20% which is comparable with various other studies.

We followed the case for 2 weeks after diagnosis. Out of 32 alive cases 12 had recovered completely and the other 20 had residual weakness. Since the patients were not followed up for longer duration we could not comment on long term outcome of them.

Contrary to ischemic arterial stroke, CVT could be described as an "all or nothing" disease with good short and long term outcomes when the acute phase of illness has been survived[85].

CONCLUSION

CONCLUSION

1. The present study emphasizes that cerebral venous thrombosis is an important cause of stroke especially in the peripartum settings and stroke in young.
2. The mode of onset is variable and spectrum of clinical presentation is extremely wide.
3. Diagnosis of CVT needs high index of suspicion.
4. Demonstration of underlying etiology may be difficult if it is not clinically evident.
5. Prompt diagnosis and anticoagulation affects patients outcome.
6. Imaging plays a key role in diagnosing cerebral venous thrombosis, a condition that can be mimicked by several other neurological entities.
7. The most sensitive diagnostic modality of choice is MRI with MR venography.
8. Multiple risk factors can be present in a single patient.
9. Anticoagulants is appropriate and the prognosis is generally favorable.
10. Systematic workup of hereditary thrombophilic conditions can be done wherever possible to find out the treatable cause.

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ANNEXURE

- **Proforma**
- **Master Chart**
- **Ethical Committee Approval Order**
- **Consent Form**

PROFORMA

NAME: DOA:

AGE: DOD/E:

SEX: male / female Hospital No:

Occupation: Address:

Socio-economic status: Literacy status:

COMPLAINTS:

1. Head ache: yes/no a) Type: frontal/temporal/occipital/diffuse

b) Side: right/left/bilateral

c) Duration:

2. Convulsions: yes/no a) focal/GTCS/focal +GTCS

b) single/status/multiple

c) Duration:

3. Limb weakness: yes/no right/left/both

4. Consciousness: normal/altered

5. Speech disturbances: normal/aphasic

6. Visual disturbances: yes/ no

7. Chronoloogy of symptoms: static/progressive/regressive/fluctuating

8. Neuro psychiatric illness:

9. Non-neurologic symptoms: yes/no

Fever

Vomiting

Diarrhea

Ear ache/ discharge

10. Rheumatological symptoms: yes/no

Arthralgia

Oral ulcers

Skin rashes

Alopecia

Photosensitivity

Others

PAST HISTORY:

DM/SHT/TB/CAD/OCP intake/similar illness/epileptic

PERSONAL HISTORY:

1. Smoker yes/no 2. Alcohol yes/no

3. I.V drug abuse yes/no 4. Pan user yes/no

MENSTRUAL HISTORY: (If female)

Regularity: Married: yes/no Abortions:

IF POSTPARTUM:

a. Duration of pregnancy at delivery

Abortion/Pre term/Full term/Post term

b. Place of delivery: Home / Hospital

c. Type of delivery: Normal / Caesarean

d. Antecedent events: Present / Absent

If present, Pre-eclampsia/APH/PPH/Retained placenta

e. Duration between delivery and onset of symptoms

< 24 hr/1-10 days/11-20 days/20-30 days/>30 days.

FAMILY HISTORY: DM/ SHT/ stroke/ rheumatologic illness.

CLINICAL FINDINGS:

Pulse BP temp

Pallor: Present / Absent

Icterus: Present / Absent

Cyanosis: Present / Absent

Clubbing: Present / Absent

Pedal edema: Present / Absent

Lymphadenopathy : Present/Absent

Signs of dehydration:

SYSTEMIC EXAMINATION:

Neurological Examination:

1. Glasgow Coma Scale
2. Level of consciousness: Conscious/Drowsy/Stuporus/Coma
3. Speech: Normal/Aphasia
4. Fundi abnormalities: RT LT (Normal/ Papilledema)
5. Gaze paresis
6. Cranial nerve palsies
7. Tone: RT LT (Normal/Increased/ Decreased)

   UL

   LL
8. Power: RT LT (Normal/ Decreased)

   UL

   LL
9. Deep tendon reflexes: RT LT (Normal/Brisk/Sluggish)

   UL

   LL
10. Plantar reflex: RT LT (Flexor/Extensor/Equivocal)
11. Sensory system: RT LT (Normal/Decreased/Cannot be assessed)
12. Cerebellar system: RT LT (Present/Absent/Cannot be tested)
13. Extra pyramidal involvement: Present / Absent
13. Gait abnormalities: Present / Absent

14. Bladder involvement: Present / Absent

15. Signs of meningeal irritation: Present / Absent

CVS: Normal/Abnormal

RS: Normal/Abnormal

P/A: Normal/Abnormal

ENT: 1. Sinusitis - Present / Absent 2.Mastoiditis - Present / Absent

INVESTIGATIONS

Hb: TC: DC: N L M E B ESR: Platelets:

P/S:

BT: CT: PT: INR: APTT:

RBS:

T.Cholesterol:

LDL:

TGL:

Serum urea:

Serum creatinine:

Serum sodium:

Serum potassium:

Serum bilirubin:

ECG:

Urine routine:

Chest X-ray PA view:

CT:

MRV:

ANA (if done):

APLA (if done):

RA factor (if done)

TREATMENT DETAILS:

Anti coagulants:

Anti edema measures:

Anti epileptics:

Physiotherapy:

OUTCOME:

Improved/ Static/ Deteriorated/Expired.

(42)

INSTITUTIONAL ETHICAL COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-600 003

Telephone 25363970
Fax 044 2535115
Dated : 12.05.2010

L.Dis.No.14597/ME5/Ethics Dean/MMC/2010

Title of the work : "A clinical study of cerebral venous thrombosis."

Principal Investigator : Dr. B. Priyadharsini
Designation : PG in MD General Medicine
Department : Madras Medical College & GGH, Ch-3.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 12th May 2010 at 2.p.m in Pharmacology Seminar Hall, Madras Medical College, Chennai -3

The members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
3. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
4. You should not deviate form the area of the work for which you applied for ethical clearance.
5. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
6. You should abide to the rules and regulation of the institution(s).
7. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
8. You should submit the summary of the work to the ethical committee on completion of the work.
9. You should not claim funds from the Institution while doing the work or on completion.
10. You should understand that the members of IEC have the right to monitor the work with prior intimation.

SECRETARY
IEC, MMC, CHENNAI

CHAIRMAN
IEC, MMC, CHENNAI

DEAN
MADRAS MEDICAL COLLEGE,
CHENNAI -3

PATIENT CONSENT FORM

Title	:	Anemia in post renal transplant patients
Study centre	:	Govt.general hospital, MMC,chennai
Patient's name	:	
Patient's age	:	
Identification number	:	

I confirm that I have understood the purpose of the procedures of the above study. I have had the opportunity to ask questions and all my questions have been answered satisfactorily. ☐

I understand that my participation in the study is entirely voluntary and that I am free to withdraw from the study at any time without my legal rights being affected ☐

I understand that sponsors of the study, others working on sponsor's behalf, the ethics committee and the regulatory authorities

will not need my permission to look at my health records both in

respect of current study and any future research that may be conducted ☐

in relation to it; even if I withdraw from the study I agree to this access. However I understand that my identity will not be revealed in any information released to third parties unless required by law. I agree not to restrict the use of any data or results arising from the study.

I agree to take part in the above the study an to comply with the instructions given during the study and inform about any change in my health status to the investigator. ☐

I hereby give permission to undergo complete clinical examination and investigations as part of the study. ☐

Signature of the patient

Patient's name and address

place

date

Signature of the investigator

Investigator's name

place

date

MASTER CHART

NAME	AGE	SEX	ONSET	P / NP	COMPLAINTS									SIGNS								OUTCOME
					HEADACHE	FOCAL DEFICI	SEIZURE	CONSCIOUSNE	FEVER	EAR DISCHAG	DIARROHEA	COMORBID STATU	DRUG ABUSE	PALLOR	VITALS	HEMIPLEGIA	CRANIAL NER	NECK STIFFNE		FUNDUS	OTHER SYSTE	
1. AMUDHA	22	F	SA	P	+	-	-	D	+	-	-	PIH	-	+++	S	-	9,10	+		PE	-	A
2. JOTHI RAMAN	29	M	SA	NP	+	+	FO/GT	CO	-	-	-	INS	S/A	-	US	LH	-	+		PE	-	D
3. SWATHY	25	F	SA	P	+	-	GT	S	+	-	-	SEI	-	+	S	-	-	-		-	-	A
4. BABU	21	M	A	NP	+	+	GT	C	-	-	-	-	A	-	S	LH	-	-		DB	-	A
5. PRABHU	31	M	SA	NP	+	+	GT	S	+	-	-	HT	S/A	-	US	RH	6	+		PE	-	A
6. KARTHIKEYAN	28	M	CH	NP	+	-	-	C	+	-	-	HIV	PAN	-	S	-		-		N	-	D
7. SATHYA	34	M	SA	NP	+	+	FO/GT	D	-	-	-	-	S/A	-	S	LH	-	+		DB	11	A
8. RAJESHVARI	55	F	A	NP	-	+	-	C	-	-	+	HT/DM	-	+	S	RH	-	-		-	-	A
9. CHANDRAN	40	M	SA	NP	+	+	GT	D	-	-	-	-	S/A	-	S	LH	3	-		-	-	A
10. GURU	27	M	A	NP	+	-	-	C	+	-	-	TB	S/A	-	S	-	6	+		PE	RALES	A
11. NATHAN	32	M	SA	NP	-	+	FO/GT	S	+	+	-	CSOM	-	++	S	RH	8	-		-	CSOM	A
12. SANGEETHA	20	F	A	P	+	-	GT	S	-	-	-	-	-	+	S	-	6	+		PE	-	A
13.PRAKASH	28	M	SA	NP	+	+	SE	CO	-	-	-	BA	S/A	-	US	FB	6	+		PE	WHEEZ	D
14.MOHAN	42	M	SA	NP	+	+	GT	D	-	-	-	HT	S	-	S	LH	-	-		-	-	A
15. ABRAHAM	40	M	A	NP	+	+	FO/GT	D	+	-	-	-	S/A	-	US	RH	6	+		PE	-	D
16. KUMAR	36	M	SA	NP	+	-	GT	S	+	-	-	-	-	+	US	-	-	+		DB	-	A
17. AMULU	23	F	SA	P	+	-	SE	CO	-	-	-	-	-	++	US	-	7	-		-	-	D
18. CHITHRA	26	F	A	P	+	-	GT	D	-	-	-	-	-	++	S	-	-	-		-	-	A
19. MURUGAN	31	M	CH	NP	+	+	FO	C	-	-	-	APLA	PAN	+		RH	-	-		DB	-	A
20. PONNI	25	F	SA	P	+	-	FO	C	+	-	-	RHD	-	+		-	-	-		DB	MDM	A

MASTER CHART

NAME	AGE	SEX	ONSET	P / NP	COMPLAINTS									SIGNS								OUTCOME
					HEADACHE	FOCAL DEFICIT	SEIZURE	CONSCIOUSNES	FEVER	EAR DISCHAGE	DIARROHEA	COMORBID STATU	DRUG ABUSE	PALLOR	VITALS	HEMIPLEGIA	CRANIAL NERV	NECK STIFFNES		FUNDUS	OTHER SYSTEM	
21.MANOJ	20	M	SA	NP	-	-	-	C	-	-	-	-	PAN	-		-	-	-		-	-	A
22.SHANTHI	22	F	SA	P	+	+	GT	D	+	-	-	-	-	++		RH	-	+		DB	-	A
23.KAVITHA	21	F	A	P	+	-	FO/GT	S	+	-	-	-	-	++		-	-	-		DB	-	A
24.MURUGAN	27	M	SA	NP	+	+	GT	D	-	-	-	TB	S	-		LH	7,9,10	-		-	RALES	A
25. STALIN	32	M	SA	NP		+	FO	C	-	-	-	-	S/A	-		LH	-	-		-	-	A
26. VASANTHA	43	F	CH	NP	-	-	SE	S	+	-	-	DM	-	-		-	6	+		PE	-	A
27. MANJU	27	F	SA	P	+	+	GT	D	+	-	-	-	PAN	-		LH	7	-		-	-	A
28. SABARI	26	M	A	NP	+	+	-	C	-	-	-	-	-	-		RH	-	-		-	-	A
29. VASU	33	M	SA	NP	+	+	GT	CO	+	-	-	HIV	PAN	-		RH	-	+		DB	-	D
30. BAVANI	25	F	A	P	+	-	-	C	-	-	-	-	-	+		-	-	-		-	-	A
31.NAMADEVAN	30	M	SA	NP	+	+	FO	C	-	-	-	-	S	-		LH	-	-		DB	-	A
32.SAROJA	24	F	A	P	+	-	FO/GT	CO	-	-	-	PIH	-	+		-	-	-		-	-	D
33. KRISHNAMURT	38	M	SA	NP	+	+	GT	D	-	-	-	-	S/A	-		FB	-	-		-	-	A
34. SHAH	41	M	SA	NP	+	+	FO/GT	D	+	-	-	HT	S	-		RH	3	+		DB	-	A
35. BALAN	36	M	A	NP	+	+	GT	D	+	-	-	BA	S/A	-		LH	-	-		-	WHEEZ	A
36. VADIVU	26	F	SA	P	+	-	FO	C	-	-	-	-	-	++		-	-	-		-	-	A
37. YASODA	29	F	SA	P	+	-	GT	CO	+	-	-	-	-	+		-	-	-		DB	-	D
38. MURUGAMMAI	22	F	A	P	+	-	-	C	-	-	-	-	-	+		-	-	-		-	-	A
39.SIVARAMAN	36	M	SA	NP	+	+	FO/GT	S	+	-	-	CSOM	PAN	-		LH	8	+		DB	CSOM	A
40. PITCHAI	29	M	SA	NP	+	-	FO	C	-	-	-	-	S/A	-		-	-	-		-	-	A

INVESTIGATIONS

NAME	SUGAR	UREA	CREATININE	LFT	HB%	PLATELETS	CSF	CT SCAN		MRI+MRV FINDINGS									PT/INR	HOMOCYSTEIN	OTHERS	OUTCOME
								DIRECT SIGNS	INDIRECT SIGNS	SSS	TS	STS	SS	CST	CV	IJV	DCV	MASTOIDITI				
1. AMUDHA	94	84	2.1	N	4	2.2	N	N	NHI	+	+		+			+		+	1.37	N	N	A
2. JOTHI RAMAN	110	24	0.7	N	12	3.2	-	EDS	HI	+	+	+							0.9	HI	N	D
3. SWATHY	100	32	0.8	N	8.2	3.1	P	N	N	+									0.92	N	ACL	A
4. BABU	98	35	0.9	N	17.2	2.6	-	CS	E	+	+								1.13	N	N	A
5. PRABHU	112	33	0.8	N	14	3.5	N		HI	+									1.21	N	-	A
6. KARTHIKEYAN	84	34	0.8	N	12	3.1	N	DTS	BHI	+									1.12	HI	-	D
7. SATHYA	111	28	0.7	N	13	2.3	-		HI		+								1.27	HI	-	A
8. RAJESHVARI	234	110	3.2	N	9	2.4	-		NHI		+								1.27	N	-	A
9. CHANDRAN	96	42	1.1	N	12	2.6	-		MS/ME	+	+		+	+					1.15	HI	-	A
10. GURU	99	32	1	N	13	2.8	p/c/x	EDS	HI		+								1.18	N	-	A
11. NATHAN	103	28	0.8	N	7.8	2.4	C		HI/E	+	+				+				1.15	HI	-	A
12. SANGEETHA	102	26	0.7	N	8	2.8	-		HI	+									1.13	N	-	A
13.PRAKASH	121	82	1.8	N	15	3.6	-		ME/E		+								1.21	HI	-	D
14.MOHAN	109	30	0.8	N	13	3.1	-		HI	+									1.12	N	-	A
15. ABRAHAM	106	28	0.7	N	14	2.6	C		MS/E		+								1.27	HI	-	D
16. KUMAR	98	26	0.7	N	10	3.4	N		HI	+									1.27	N	N	A
17. AMULU	120	32	0.9	N	7.2	2.9	-		ME/NS	+									1.37	N	N	D
18. CHITHRA	131	36	1	N	7.6	3.4	-	N	N		+								0.9	N	-	A
19. MURUGAN	85	40	1.2	N	8.4	2.4	-		HI	+									1.22	N	ACL	A
20. PONNI	76	44	1.1	N	9.6	3.1	N		HI										1.11	N	N	A

INVESTIGATIONS

NAME	SUGAR	UREA	CREATININE	LFT	HB%	PLATELETS	CSF	CT SCAN		MRI+MRV FINDINGS									PT/INR	HOMOCYSTEIN	OTHERS	OUTCOME
								DIRECT SIGNS	INDIRECT SIGNS	SSS	TS	STS	SS	CST	CV	IJV	DCV	MASTOIDITI				
21.MANOJ	75	38	1	N	14	3.8	-		HI		+								1.22	HI		A
22.SHANTHI	96	42	1.1	N	7.2	3.5	N	CS	HI	+									1.1	N	-	A
23.KAVITHA	113	36	0.9	N	7.1	3.6	-		BHI/E	+	+								1.3	N	-	A
24.MURUGAN	112	36	0.8	N	12	2.6	P/C/X		HI			+							0.9	HI	-	A
25. STALIN	86	28	0.7	N	14	3.8	-	CS	HI	+									1.25	HI	-	A
26. VASANTHA	116	44	1.1	N	13	3.1	C		HI		+								1.1	N	-	A
27. MANJU	110	38	0.9	N	12	2.9	C		HI	+									1.21	N	-	A
28. SABARI	87	36	0.8	N	13	3.4	-		HI		+								1.1	N	-	A
29. VASU	99	48	1.2	N	13	1.9	N		ME/E	+									0.9	HI	-	D
30. BAVANI	82	42	1.1	N	9.4	1.9	-	EDS	HI	+									0.9	N	N	A
31.NAMADEVAN	93	40	1	N	13	3.2	-		HI		+								1.13	N	-	A
32.SAROJA	81	96	2.8	N	9.8	2.3	-		MS/E	+	+								1.21	HI	-	D
33. KRISHNAMURTHI	79	26	0.7	N	14	2.7	-		NHI		+	+							1.12	HI	-	A
34. SHAH	115	46	1.2	N	13	2.4	P	N	N					+					1.27	HI	-	A
35. BALAN	114	36	0.8	N	12	2.9	P		HI	+									1.27	N	-	A
36. VADIVU	73	28	0.7	N	7.8	2.6	-		HI		+								1.15	N	N	A
37. YASODA	74	40	0.9	N	9.2	3.5	N	CS	ME/MS	+									1.18	HI	-	D
38. MURUGAMMAL	82	72	1.7	N	9.4	2.6	-		HI	+									1.15	N	-	A
39.SIVARAMAN	118	28	0.7	N	12	3.1	P		BHI/E		+				+			+	1.1	HI	C/S	A
40. PITCHAI	86	36	0.9	N	14	3.4	-		HI	+									0.9	HI	-	A